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A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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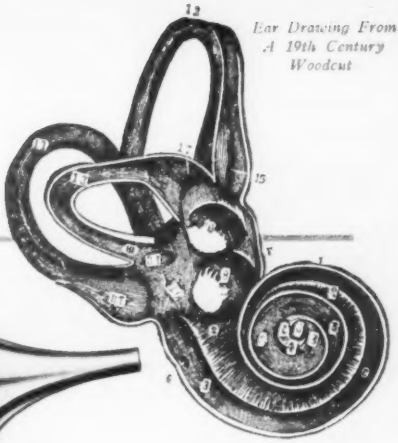

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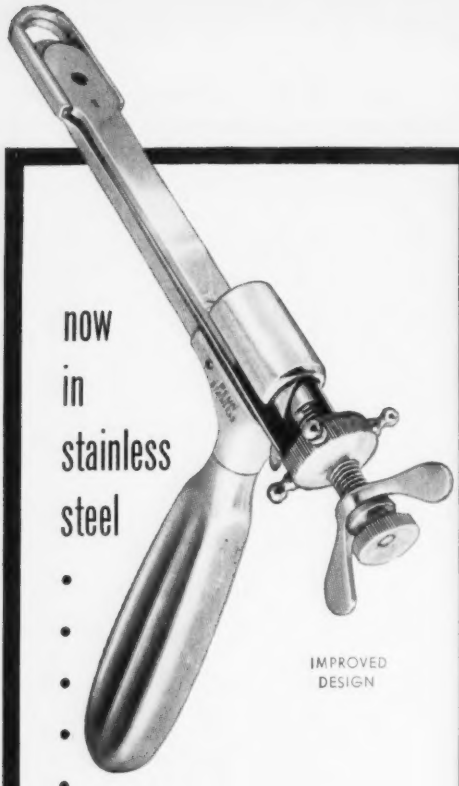

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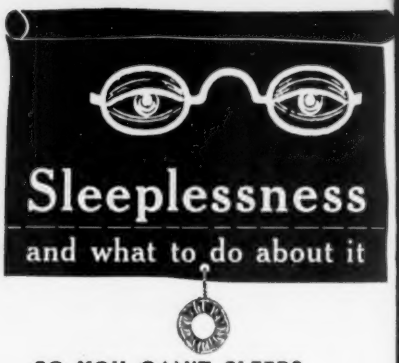

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The Changing Picture of Staphylococcic Infections in Otolaryngologic Practice

C. J. HEINBERG, M.D., Pensacola, Fla.

Introduction

The Staphylococcus (Micrococcus) Pyogenes var. aureus is probably one of the very earliest infection agents known to man, as its ravages, in the form of osteomyelitis lesions, have been recorded in bones recovered from excavations of tombs dating back many centuries in antiquity. It is likewise one of the most ubiquitous organisms, and its adaptability to environmental circumstances explains its continuing importance as a serious contender for the title of today's "Captain of the Men of Death," as pointed out recently in an editorial in the *British Medical Journal*.[1]

One can truthfully say that the first half of the 20th century has been the period in medical history which undoubtedly will be credited with medicine's greatest advance in the battle against infectious disease. It has been the period of antisepsis, asepsis, and the beginning of the antibiotic and chemotherapeutic era. But it must be confessed the final answer to the conquest of disease germs has not yet been written, as evidenced by the emergence of strains of organisms resistant to all these newer "miracle" drugs which held out so much hope when they were first introduced. And it is the Staphylococcus which heads the procession in this respect.

Otolaryngologists are perhaps even more conscious of this problem than the average physician, for their practice is concerned to a very large extent with infections of the ears, nose, nasopharynx, accessory nasal sinuses, and throat. There has been and there still is a continuing shift in the bacteria recovered from these patients. Where formerly streptococci predominated, today staphylococci are found to be the most frequently encountered and often the sole causative organism.

Historical Note

Small coccoid organisms, which in the light of our present knowledge were presumably staphylococci, were seen and even cultivated by several of the early bacteriologists after the discovery of the microscope. But it was Ogston, in 1880, who first applied the name staphylococci (derived from the Greek word "staphule," meaning grape) to them because of the resemblance of their clusters to a bunch of grapes. By the turn of the century it was recognized that the staphylococci were capable of producing toxic substances but varying greatly in degree. However bacteriologic discoveries with the identification of the causative organism in many diseases were being made at such a rapid rate during that

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period that relatively little attention was paid to this very important point until comparatively recently. But in 1929 a relatively obscure bacteriologist in a London hospital, Alexander Fleming, noted the antibacterial effect of a contaminating mold, Penicillium notatum, upon a plate culture of staphylococci, and the humble, ubiquitous Staphylococcus played its important role in the development of today's antibiotic and chemotherapeutic era.

As stated above, the staphylococci are Gram-positive spherical cells usually occurring in readily identifiable clumps. They are for the most part aerobic, or facultatively anaerobic. There are two major groups. The first, or saprophytic, variety has colonies which are usually white or pale yellow when grown in solid media; it produces several toxins, and many of its members are coagulase-positive. For purposes of convenience the term Staphylococcus will be used in this discussion to designate the pathogenic varieties encountered in clinical practice.

In this paper we are not concerned with the saprophytic forms which are present almost everywhere in man's environment. Nor do we view with too much interest those relatively benign varieties found almost normally in the average bacterial flora of the skin and nasopharynx. But we are concerned with the potentially pathogenic species of staphylococci which are being carried constantly on the skin or in the nasopharynx by approximately 50% of the population at one time or another during the year, and which may give rise to spontaneous infection of very serious possible consequence.

Staphylococcal Infection in Man

Constitutional and other factors affect markedly the susceptibility of man to infection by the Staphylococcus. That age is of importance is evidenced by the fact that while staphylococcal infections may occur in persons of all ages, they are frequently encountered in children and young adults. Acute osteomyelitis, due to the staphylococcus, is characteristically found during the first two decades of life, and secondary cutaneous infections, such as those associated with acne, tend to coincide with the physiologic changes of puberty and adolescence. Malnutrition, metabolic disorders, such as diabetes, and concurrent infection by other organisms, all of which tend to lower the general body defense mechanisms, appear to enhance the susceptibility of the individual to invasion by the Staphylococcus, especially in the older age group. Gross injury or even the minimal amount of trauma induced by friction or scratching may contribute to produce conditions favoring the development of a localized cutaneous infection by lowering local tissue resistance.

I feel sure there is general agreement that the most frequent portal of entry of the Staphylococcus to the body is the skin; in fact the majority of staphylococcal septicemias and infections of the deeper tissues have their origin in cutaneous infections. Among the common infections of the skin and subcutaneous tissues due to staphylococci are furuncles, carbuncles, folliculitis, sycosis, and paronychia, as well as most of the suppurative infections associated with wounds. Acne is more often caused by the milder Staphylococcus albus, frequently found in combination with a diphtheroid bacillus.

Furuncles, or boils arising usually in a hair follicle, occur singly or as multiple lesions which develop simultaneously or in crops. Carbuncles are caused by the infection spreading in the deeper layers of the skin and the development of multiple openings through follicles or sebaceous glands to permit the discharge of pus. Fever and malaise often accompany the clinical course of a carbuncle. Cutaneous staphylococci are found in the majority of suppurating wounds, either as the primary infecting agent or in combination with other bacteria.

Invasion of the blood stream by staphylococci results in a generalized infection which assumes either of two clinical courses

Fig. 1.—Osteomyelitis of right maxilla.

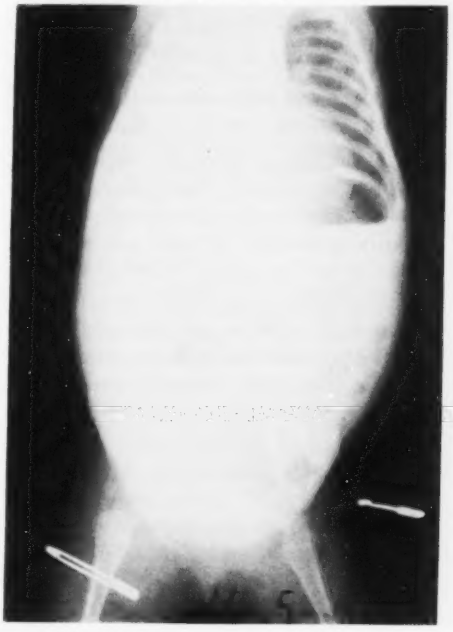
unless controlled promptly by the use of antibiotics or other chemotherapeutic agents. In the first type of reaction a fulminating systemic infection may occur, which, it should be emphasized, is fully as severe as the septicemia caused by the β-hemolytic Streptococcus. It is characterized by sustained high temperatures above 101 F, a pulse rate of 140 or more, leukocytosis, and profound toxemia, frequently associated with symptoms of irritation of the central nervous system and the gastrointestinal tract. The course is rapid, and death commonly ensues within a few days.

The second and more usual form of such systemic infection runs a more prolonged course and is typically accompanied by the development of metastatic abscesses, often multiple, and commonly found in the lungs, kidneys, heart, or bones. I wish to stress the last, inasmuch as osteomyelitis of the bones of the skull is becoming more and more an otolaryngeal complication of interest, particularly in the infant (Fig. 1).

Staphylococcal septicemia originates by invasion of the blood stream from a suppurative focus. In the case of a cutaneous lesion, anatomic features at the site of the infection sometimes determine whether a septicemia will develop. For example, should staphylococci enter the rich venous plexus in the region of the nose and upper lip as a result of trauma to a furuncle, septicemia almost invariably results in association with cavernous sinus thrombosis. Several instances of this type of infection have been observed in prize fighters receiving blows on an infected vestibule and in other persons from squeezing pustules.

Systemic infection also may arise secondarily from metastatic lesions. It is common to find a suppurative phlebitis or thrombophlebitis of the major veins in the immediate vicinity of a suppurative lesion, arising by extension from the localized infection. The intravascular lesion appears to be an important point of origin of systemic infections which arise from metastatic abscesses. Staphylococci are shed into the circulation from the surrounding inflammatory zone of the phlebitis or are carried

Fig. 2.—Staphylococcal pneumonitis.



in fragments of an infected thrombus, to lodge and establish metastases at other sites. The mortality rate in systemic infections is high, being variously reported as from 50% to 90%. A fatal outcome may be governed in large part by the extent of metastatic involvement of vital organs and tissues and their accessibility to surgical drainage. At least 90% of all cases of osteomyelitis are caused by the Staphylococcus developing by metastasis from a cutaneous lesion. The case shown in the photograph (Fig. 1) represents a primary infection of the lachrymal sac which extended to the maxilla with the development of an osteomyelitis and spread from there by the blood stream to the lung to produce a secondary pneumonitis (Fig. 2).

Staphylococcal infection of the lungs is represented by clinical forms varying from mild tracheobronchitis to acute bronchopneumonia and the formation of multiple abscesses. Approximately 9% of bronchopneumonias are due to staphylococci, the mortality rate often being high. Acute staphylococcal pneumonia is believed to originate by direct extension from the upper respiratory tract, either by inhalation or by lymphatic spread. These infections are seen chiefly in the very young and the very old.

Staphylococcal Infection in the Hospital

In the past two or three years there has been a marked increase in staphylococcic infections in hospitalized patients. This has assumed increasingly serious and alarming proportions in many general, and especially in pediatric, hospitals. These institutions have been accused of laxity in asepsis and too much dependence on antibiotics. Particularly in pediatric nurseries outbreaks of epidemic proportion have occurred in the newborn. These have involved skin, nose, and throat foci and in many cases have gone on to the development of metastatic pneumonia.

In Australia the investigation of a number of such outbreaks by the Fairfax Institute of Pathology uncovered the fact that many persons on the nursing and medical staffs were nasal carriers of S. aureus, and that, in spite of rigid precautions in the nurseries, infections of the newborn continued as long as those carriers were in contact with the babies. It is interesting to note that 73 out of 101 strains isolated from the professional staff were penicillin resistant. Antibiotic nasal ointment was used to treat these carriers, and there was a resultant drop in the carrier rate, with a simultaneous reduction in the neonatal infections with staphylococci.

In addition to rigid asepsis in the operating room and in handling surgical patients, prophylactic measures were applied to the professional personnel as well as to the physical plant at the Veterans' Administration Hospital at Manchester, N. H. Cultures from the throat and nares of the entire staff were taken, and, if found positive for staphylococci, treatment was required until cultures became negative. The hands and forearms of aides and nurses were inspected carefully before they reported to duty and those with any suspicious lesions were checked by a staff physician.[4]

Since the anatomical area involved in many of these primary staphylococcic infections represents the field of especial interest to the otolaryngologist, it is incumbent upon him to be aware of the potential dangers of such infections. He should take immediate and adequate steps to abort these foci of infection and prevent the occurrence of metastatic lesions if possible. Too much confidence has been placed on the routine administration of penicillin or one of the broad-spectrum antibiotics without bacterial sensitization tests. The progressive curtailment of prophylactic and therapeutic effectiveness of penicillin in hemolytic S. aureus infections shows that a progressively large number of strains of this organism have acquired more or less permanent resistance to this antibiotic agent. Altemeier[5] tested, in vitro, 1422 consecutive strains of hemolytic S. aureus isolated from surgical infections and studied at the Research Surgical Bacteriological Laboratories of the

University of Cincinnati over a five-year period. These revealed wide variations in the percentages of penicillin-resistant strains examined at three-month periods. The variations ranged from 19% to 80%, and suggested that cyclic variations may occur at intervals of several years. However, the general trend was for an over-all progressive increase in resistant strains. This resistance feature did not prove to be so marked with oxytetracycline. It was stated by the author that unknown factors are at work that may either cause irregular spread of penicillin-resistant strains or produce cyclic variations in the incidence of acquired penicillin resistance.

Penicillin resistance[6] in staphylococci is unquestionably an extremely complex mechanism and cannot be analyzed solely on the basis of genetics or mutation of the strains of staphylococci. There is a steadily increasing array of evidence to show that the intensive use of penicillin and, to a less degree, most of the other antibiotics in an institution is followed by an increased incidence of staphylococcal infections, invariably due to resistant strains of the organism. Finland, in a series of papers covering over a five-year period from the Boston City Hospital, has shown the same emergence of resistant forms of staphylococci not only to penicillin but to practically all the other antibiotics.

Extensive studies on the flora of the air revealed that organisms of the Micrococcus group are able to survive for long periods in the air and dust in a dried state. These studies also demonstrated that microbial populations in hospitals contain more pathogens and potential pathogens than nonhospital and outdoor areas. The antibiotic resistance of air-borne staphylococci from the hospital wards and other areas where antibiotics were used with any frequency as compared with those obtained from outside sources showed a pattern of increasing resistance in relation to the therapeutic index.[7,8] It is noteworthy that the greatest resistance was found in organisms from the pediatric and surgical wards, where antibiotics were more extensively used.[9]

In our own cases the value of bacterial sensitization tests was indisputable and demonstrated that the S. aureus varies tremendously in its sensitivity to various antibiotics, especially when associated with mixed infections. The source of the strain also seemed to modify the resistance. Private patients with initial staphylococcal infections rarely harbor a particularly resistant strain, whereas strains from hospitalized patients commonly show a high degree of resistance to penicillin.

The accompanying table (Table 1) illustrates that the S. aureus from hospitalized cases is resistant to penicillin in some degree in the majority of instances, regardless of the site from which it was obtained.

Four illustrative cases are presented, three of them with pure cultures of S. aureus, the fourth with an α-Streptococcus as a secondary complicating factor. Three of the staphylococci strains recovered culturally were penicillin resistant, and successful treatment could be attributed in large measure to recognition of this fact and the use of therapeutic agents other than penicillin.

CASE 1.—Newborn infant. On the third day after birth developed a purulent conjunctivitis in the left eye. On the fourth day became febrile. The following day cough developed, and otolaryngological consultation was requested. X-ray (portable) demonstrated osteomyelitis of the left maxilla and secondary pneumonitis. The laboratory reported pure culture of S. aureus, which was coagulase-positive. Antibiotic sensitivity tests showed 4+ sensitivity to all antibiotics except oxytetracycline (Terramycin), to which the organism was 3+ sensitive. Treatment with penicillin and suspension of erythromycin (Erythrocin) was instituted. Aerosol Alevaire (an alkylaryl polyether alcohol detergent) to which was added soluble penicillin was given by means of tent. Gradual resolution of the infection followed, with all traces of the osteomyelitis and pneumonitis clearing in three weeks.

CASE 2.—Man. Purulent drainage of the right mandible for four weeks subsequent to dental extraction. Had been given penicillin with no results. X-ray demonstrated osteomyelitis of the mandible. The drainage was cultured, and the

TABLE 1.—*Staphylococcus Aureus Sensitivity in Twenty-Seven Cases of Otolaryngologic Infections*

Case No.	Clinical Diagnosis	Source	Secondary Organisms Encountered on Culture	Penicillin	Streptomycin	Oxytetracycline	Tetracycline	Chlortetracycline	Erythromycin	Chloramphenicol	Nitrofurantoin	Trisulfapyrimidines	Bacitracin	Albamycin	Novobiocin	Mysteclin *	Neomycin
1	Furunculosis vestibule	Nose	None	R	R	R	R	R	4	4	4	R	—	—	—	—	—
2	Third-degree burn	Face	Aerobacter	R	1	2	2	R	R	1	R	R	—	—	—	—	—
3	Pharyngitis granulosa	Throat	Neisseria	R	R	R	R	R	4	4	4	R	—	—	—	—	—
4	Acute follicular tonsillitis	Tonsils	Nonhemolytic Streptococcus	4	4	2	2	2	2	4	4	R	—	—	—	—	—
5	Nasopharyngitis	Pharynx	Streptococcus	4	4	2	2	2	4	4	4	R	—	—	—	—	—
6	Pharyngitis	Throat	S. albus	4	4	4	2	3	4	4	R	R	—	—	—	—	—
7	Cellulitis with abscess	Neck	None	R	R	R	R	R	4	4	4	R	—	—	—	—	—
8	Furuncle	Face	None	R	4	R	R	R	4	4	4	R	—	—	4	R	—
9	Pharyngitis	Throat	S. albus Streptococcus	R	R	R	R	R	4	4	4	R	—	—	—	—	—
10	Tracheobronchitis	Sputum	Heavy α-Streptococcus	R	2	2	2	2	4	4	R	R	—	1	—	1	2
11	Peritonsillar abscess	Pus	None	R	R	R	R	R	3	4	3	R	—	4	4	R	—
12	Furunculosis	Scalp	None	R	R	R	R	R	3	4	3	R	—	4	4	R	—
13	Maxillary sinusitis	Nasal disch.	α-Streptococcus	4	4	R	R	3	4	4	4	R	—	—	—	—	—
14	Tonsillopharyngitis	Throat	α-Streptococcus; Neisseria	4	4	2	R	3	4	4	3	R	—	R	2	3	—
15	Carbuncle	Pus	None	4	2	3	3	3	3	2	3	R	—	3	3	4	—
16	Acute follicular tonsillitis	Throat	S. albus Streptococcus	R	R	R	R	R	3	4	3	R	—	2	4	R	—
17	Nasopharyngitis	Nose & throat	α-Streptococcus; Neisseria	R	R	2	2	2	R	2	4	R	—	—	—	4	—
18	Pharyngitis	Throat	α-Streptococcus	4	2	3	3	2	4	4	4	R	—	3	—	3	—
19	Acute tonsillitis	Throat	S. albus	4	R	R	R	3	R	R	4	R	—	—	—	—	—
20	Osteomyelitis mandible	Drainage	None	R	R	R	R	R	R	3	4	R	—	—	3	R	—
21	Acute otitis media	Drainage	None	2	2	R	R	2	3	3	3	R	—	3	3	R	—
22	Catarrhal nasopharynx; purulent otitis media	Throat & Ear	Neisseria & α-Streptococcus	4	2	R	R	2	2	2	3	R	—	2	3	R	—
23	Bilateral ethmomaxillary sinusitis pharyngitis	Nose & throat	α-Streptococcus	R	R	R	R	R	2	3	4	R	—	2	3	R	—
24	Tracheobronchitis	Aspirated sputum	None	R	R	R	R	2	R	3	2	R	—	2	3	R	—
25	Osteomyelitis maxilla	Purulent exudate	None	4	4	3	4	4	4	4	4	4	—	4	—	4	—
26	Otitis externa furunculosis	Ear	None	R	2	R	R	R	4	4	3	R	—	2	—	1	—
27	Adenotonsillitis	Throat	α-Hemolytic Streptococcus	4	4	2	R	2	3	4	4	R	—	—	—	—	—

Key: 4—very sensitive; 3—moderately sensitive; 2—slightly sensitive; 1—sensitivity borderline. R—resistant.

Interpretation: Antibiotic sensitivity tests were read at lowest zone of sensitivity for Staphylococcus aureus. Sensitivities of the secondary organisms were disregarded and are not shown in this table.

Sensitization tests performed in the laboratories of Sacred Heart Hospital under the direction of Dr. G. Hilbert, Head of the Department of Pathology and Bacteriology.

* A combination of tetracycline and nystatin (Mycostatin).

laboratory reported a pure culture of S. aureus, which was coagulase-positive. Sensitivity tests evidenced a resistance to all antibiotics except nitrofurantoin (Furadantin) and a 3+ sensitivity to chloramphenicol (Chloromycetin). Treatment consisting of curettement of the area involved with administration of nitrofurantoin caused an uneventful recovery.

CASE 3.—White woman, 18 years of age. Complained of purulent discharge from the left ear for six weeks. Had earache after swimming, and tympanotomy was done by her otologist, with subsequent drainage. The usual cleansing and ear drops failed to cause a cessation of the discharge. The laboratory reported a coagulase-positive S. aureus in pure culture. Sensitivity tests showed the organism to be only slightly sensitive to penicillin and 3+ sensitive to erythromycin, chloramphenicol, nitrofurantoin, and novobiocin (Albamycin). Treatment consisted of nasopharyngeal asepsis, erythromycin systemically, and cleansing of the canal with insufflation of powdered nitrofurantoin. The discharge gradually subsided until the ear became dry. The perforation of the tympanic membrane was then cauterized with trichloracetic acid around the margins, and a "fish skin" placed over the perforation. Healing occurred in three weeks.

CASE 4.—White girl, age 6 years. Sudden onset of fever and cough. Dyspnea. Diagnosis of acute tracheobronchitis. Direct laryngoscopy with aspiration of sputum secretions. Laboratory reported coagulase-positive S. aureus mixed with heavy α-Streptococcus. Antibiotic sensitivity tests showed a 4+ sensitivity to erythromycin and chloramphenicol. The α-Streptococcus was highly sensitive to penicillin. The child was placed in an oxygen tent in which cold oxygen and aerosol Alevaire with soluble penicillin was administered. Pediatric suspension of erythromycin was given in maximum dosage. A tracheotomy set was kept by the bed so that, should the dyspnea increase and cyanosis develop, the patient could have an emergency procedure in an adjoining surgical cubicle. Immediately after the foregoing treatment was instituted the process began to subside, and after a night of watchfulness the patient was much improved. In 48 hours the child was discharged from the hospital as cured.

This emphasized the futility of blindly giving patients penicillin for these infections. If we do not block and eradicate foci of hemolytic S. aureus in the otolaryngologic field by the use of the antibiotic to which its greatest sensitivity can be demonstrated, we are inviting disaster in the form of metastatic lesions which can even go on to a fatal outcome. This is particularly important in infants, in whom a simple infection can be causal of osteomyelitis, pneumonitis, or metastatic abscesses in remote viscera. It is difficult to understand why separate cultures from the nose and throat, apparently from the same initial infection, sometimes will show marked variation in their sensitivity to penicillin. Since spontaneous infections in man are known to increase immune substances in the blood, it might be rationalized that the circulating antibodies vary both in amount and effectiveness and therefore virulence is affected (Table 2).

An important problem in laboratory diagnosis is the differentiation of pathogenic from nonpathogenic staphylococci. In the past emphasis has been given to pigmentation, hemolysis on blood-agar, fermentation of mannitol, and liquefaction of gelatin. Special culture media for demonstrating these reactions have been proposed for the isolation and identification of pathogenic staphylococci.[10] There is little question that freshly isolated staphylococci from pathologic sources tend to exhibit all or most of these properties, which can thus serve as

TABLE 2.—*Summary of the Sensitivity of the Twenty-Seven Staphylococcus Aureus Strains Shown in Table 1*

	Resistant	Very Sensitive	Moderately Sensitive	Slightly Sensitive
Penicillin	16	10	0	1
Streptomycin	12	8	1	6
Oxytetracycline	16	1	7	3
Chlortetracycline	11	1	11	4
Tetracycline	18	1	6	2
Erythromycin	5	13	5	4
Chloramphenicol	1	17	6	3
Nitrofurantoin	3	18	5	1
Trisulfapyrimidines	26	1	0	0
Bacitracin	0	0	0	6
Albamycin	1	2	4	0
Novobiocin	0	4	6	1
Mysteclin	9	3	2	2
Neomycin	0	0	0	1

This table shows summary of 27 sensitivity tests on cultures taken from private patients admitted to Sacred Heart Hospital as done in the Department of Bacteriology and Pathology under the direction of Dr. G. Hilbert.

presumptive evidence of pathogenicity. However, it must be kept in mind that individual strains vary considerably and that nonpathogenic varieties exhibit some of the same reactions, although usually to a less degree. The coagulase test, on the other hand, constitutes the most reliable, single in vitro criterion for the identification of pathogenic staphylococci, and from a practical viewpoint it might be said that the cultural properties only support the evidence supplied by it.

Making use of the fact that most of the pathogenic staphylococci are capable of coagulating normal plasma, a "slide test" for their identification has been devised. It is reliable, rapid, and applicable to the routine testing of cultures and to surveys in which large numbers of colonies on plates are examined directly.[11]

It is incumbent upon the otolaryngologist therefore to first determine by proper laboratory procedures whether the infecting organism which he has recovered is coagulase positive or negative. If it is positive, it is almost mandatory to have sensitization tests done to determine to which antibiotic the particular organism is the most sensitive. This is a duty and obligation which, in too many instances, is neglected. It is to be remembered that the most innocent-appearing nose and throat infection can be the focus for fatal metastasis.

Selye [12] has suggested that stress might be a factor in these infections. In his work on the general adaptation syndrome he postulates that infections of Waldeyer's ring occurring after exposure to cold or other nonspecific injuries may be due to the lymphatic tissue undergoing acute disintegration as a result of a defensive corticoid-hormone overproduction. He further suggests that micro-organisms secondarily invade this damaged lymphatic tissue, which it has been shown represents a particularly favorable medium for microbial proliferation.

It has been previously stated that most environmental forms of staphylococci are saprophytic. Perhaps the most interesting role of STH in the adaptation syndrome is that it can effectively combat catabolism and the susceptibility to infections. Selye [13] has stated that animals heavily overdosed with corticotropin (ACTH) tend to lose a great deal of weight. Eventually they die, almost always as a result of generalized septicemia, caused by organisms normally saprophytic. In rats the lung tissue seems singularly disposed to such infections. It is therefore incumbent on otolaryngologists who keep patients on corticotropin or corticosteroid therapy for any length of time to be watchful and to give antibiotics as preventive medicine.

Corticotropin and the steroid hormones should be reserved for the treatment of diseases that may cause long or permanent incapacitation, lasting damage to vital organs, or death.[14]

Untoward reactions to corticotropin, cortisone, and analogues of these drugs may threaten life. Complications noted among children include overwhelming infection, status epilepticus, and other central nervous system disorders, gastrointestinal bleeding and perforation, severe fluid and electrolyte imbalance, spontaneous bone fracture, insulin-resistant diabetes, and protracted renal insufficiency. Intercurrent infection, the most frequent hazard of adrenal-cortical therapy, may produce no obvious manifestations until far advanced. Staphylococci immune to the usual antibiotics are often responsible.

Otolaryngologists should not use corticotropin or any form of cortisone therapy until they thoroughly familiarize themselves with the proper administration and complications of these steroids. Severe fluid imbalance and sodium retention may manifest themselves on sudden withdrawal of the drugs. The steroids have a certain and valuable place in our practice when properly administered and when one is aware of the dangerous sequelae and diligently watches for untoward reactions. Particular reference is made here to parenteral or oral ad-

ministration, as any complications from topical applications are negligible.

The use of silk as a suture material is still in vogue by many otolaryngologists. This material sometimes acts as a foreign body and results in staphylococcal abscesses. These abscesses may be resistant to large doses of antibiotics and disappear only on removal of the sutures. Bacteremia and metastatic abscess may result from suture abscesses. Such abscesses have occurred in the heart and great vessels after cardiovascular surgery. Only when the sutures were removed did the staphylococcal infections subside.

All otolaryngologic specialists are familiar with staphylococcal infections of the ear, both acute and chronic. In the external canal both furunculosis and the diffuse types are primarily staphylococcal infections. Sometimes the diffuse cases show a mixed culture of hemolytic S. aureus with Proteus vulgaris (Bacillus proteus) and Pseudomonas aeruginosa. In my own experience S. aureus, α-Streptococcus and Hemophilus influenza are the most frequent invaders in otitis media.

Goldstein [16] states that the most commonly encountered pathogens in chronic otitis media are the same that I found in otitis externa, namely the hemolytic S. aureus, P. vulgaris, and Pseudomonas. It is important to do cultures and sensitivity tests for evaluation and proper treatment of these cases. In Goldstein's series it is interesting to note that recultures of these ears while under treatment showed different organisms in 9 of 14 cases: five were of the same species, but none were of a similar strain. The bacteria either had developed resistance to the antibiotic or the sensitive strains were eliminated and the resistant strains emerged. Seven per cent of cases examined developed resistant organisms while under treatment, and the conclusion is reached that reculture is periodically necessary in refractory cases. Treatment varies with the laboratory findings, and antibiotic therapy is correlated with the laboratory sensitivity reports.

Generally speaking, the body defenses have walled off the middle and external ear in these local areas of chronic infection by sclerosis of bone and thrombosis of vessels. Saprophytic bacteria are found which live on necrotic tissue and are scavengers. Antibiotics administered systemically would bring very little of the drug to these areas. Local application of high concentrations of the antibiotic in powder form is necessary. If the infection can be controlled, the discharge eliminated, and the ear rendered dry, then surgical closure of perforations may be accomplished. If other indications of persistent infection exist, adequate aural surgery must be done.

There is normally a high occurrence of staphylococci in the nares of the average healthy population. At Ohio State University a sampling of the incidence of carriage of S. pyogenes in the nares of hospital personnel [17] revealed that in 170 persons 62, or 36.6%, had coagulase-positive staphylococci in the nose. The infant in nurseries is particularly susceptible to infection from this source by means of droplet infection. The infants in turn may transmit it to the mothers. An examination of 78 mothers on admission to the maternity ward at University Hospital in Columbus, Ohio, showed that not one of the mothers harbored the strain of staphylococci causal of an epidemic of staphylococcal infection in the nursery and among nursing mothers.

All specialists in otolaryngology are cognizant of furunculosis of the vestibule and its commonplace occurrence. These are almost invariably staphylococcal infections which, on occasion, may be mixed with streptococci. A fatal outcome has been known to result from metastasis to the brain with subsequent abscess formation. We have all seen purulent sinus infections, particularly in the frontal sinus, with osteomyelitis as a complication caused by S. aureus.

Pharyngitis, tonsillitis, and abscess, both tonsillar and retropharyngeal, have, in our experience, based on cultures, been mixed infections with S. aureus predominating but usually complicated by the presence of S. albus and α-Streptococcus.

Treatment

One might point to the treatment of staphylococcic infections over the years as representing a reasonably accurate history of the evolution of pharmaceutical and medical progress in the therapy of infectious disease generally. In superficial infections topical agents ranging from a wide variety of antiseptic inorganic preparations to the current combinations of steroids and antibiotics have been tried with varying degrees of success.

But it is the serious, systemic infections that have presented the real challenge to the chemist, biologist, and physician. It is a long cry from the incantations and potions of medieval times to the antibiotics and synthetic chemotherapeutic agents of today. Yet it is the Staphylococcus that is threatening to emerge again as the victor in spite of these newer remedies.

Today with the proper selection of therapeutic agents, whether biologic or synthetic, but based on laboratory determination of sensitivity, we can still control the vast majority of infections by these resistant staphylococci. However, the increasing number of strains resistant to some or all of the antibiotics magnifies the seriousness of the problem.

Antibiotics are by no means the first biologic approach to the control of these infections. In the earlier days of bacteriology vaccines were the most obvious method to explore. Undoubtedly some benefit has resulted from such vaccine therapy, particularly with autogenous vaccines, in the treatment of recurring cutaneous Staphylococcus infections. However, it has not fulfilled the hopes of 40 years ago. Staphylococcal vaccines often contain a small amount of toxoid, and it is not unlikely that some of the beneficial effects, both experimentally and in man, come from the antitoxic immunity which they induce.

Staphylococcic toxoid appears to be of even greater value than vaccines in the treatment of subacute or chronic cutaneous infections. The immunity derived is antitoxic; while it persists, the tissues are protected against the necrotic effects of the toxin and normal reparative processes are enhanced, but no true antibacterial effect is obtained. Staphylococcic antitoxin has been used to tide the patient over an emergency period in the past. It must not be forgotten that after the survival of the acute phase of the infection the clinical picture becomes chiefly that of the effects of metastases which are so characteristic of invasion of the blood stream and other organs by staphylococci. Antitoxin therapy cannot be expected to influence an infection due to a nontoxigenic strain.[18]

Localized staphylococcal infections appear at times to be benefited by bacteriophage therapy. Investigation of clinical applications of bacteriophages followed closely upon the discovery of the phenomenon of transmissible lysis,[19] that is, the invasion of the bacterial cell by a specific agent (bacteriophage) resulting in the disruption of the cell and the release of numerous progeny capable of repeating the invasive and disruptive process in other cells of the same species. In line with this concept Besredka postulated that if the skin were immunized it would result in immunization of the whole organism. To this end he prepared a filtrate by three successive inoculations of the pathogen until the third plate developed only a sparse growth because the pathogen developed its own antigen. The filtrate of this growth contained toxic substances which were thermolabile and a nontoxic substance which was thermostabile. Therefore by heating, the toxic portion was destroyed. In an epidemic of anthrax which destroyed many of the horses of the French cavalry, Besredka applied wet dressings of his filtrate to

the abraded skins of the animals and terminated the infections. Although his work has not been generally accepted, the same priciples of application are now being made with a phage lysate. Favorable clinical experience with such a Staphylococcus phage lysate has been reported.[19] The ideal agent for the control of Staphylococcus infections of less severity would be one which would restrain its multiplication and at the same time increase the natural immunity of the body to the organism by local densensitization. With this in mind a Staphylococcus bacteriophage lysate in aerosol form has been developed and tried clinically. By the airway route local immunity is afforded by nasal aerosol inhalation, and desensitization to the specific bacterial protein does occur to some extent. The latter is an important factor in many allergies. The rationale is similar to Mackenzie's desensitization of hay fever patients by local therapy.[20]

Baker[21] likewise claims relief of bronchial asthma, in patients where infection and bacterial allergy are believed to be contributory factors, by the use of such a lysate aerosol. In my own rather limited experience I have not been unduly impressed by the results in cases with positive sputum for staphylococci, and am inclined to feel that the use of Staphylococcus toxoid, as suggested by F. Hansel, produces equally good, if not better, results if the concentrations are kept at a well-diluted point and carefully supervised.

In fulminating infections, and those acute infections which might possibly metastasize, our treatment takes definite form today. Once the laboratory confirms the specific bacterial diagnosis from material submitted, we then are informed if the S. aureus is coagulase positive. The next step is to determine the strain and its degree of sensitization to the entire gamut of antibiotics by means of plate tests. More accurate, but more expensive and time-consuming, is the tube dilution method. The objective by either technique is to shorten the course of an acute infection and reduce the chances of complications or of fatalities. Once the antibiotic is determined, it may be administered alone or in combination with another antibiotic to which the pathogen is also sensitive. Nasal carriers must be sterilized or removed from contact with the patient, particularly in nurseries.

Probably the most recent advance in the use of antibiotics has been their supplementation with γ-globulin to provide a passive immunity while the antibiotic effect was developing. Results to date are most encouraging.

Although this series would tend to discount the value of trisulfapyrimidines (triple sulfonamides) in staphylococcal infections, there is a new sulfonamide derivative that in our experience seems to be highly effective against antibiotic-resistant staphylococci. This substance, sulfaethidole (sulfaethylthiadiazole),* has a very broad bacterial spectrum. Impressed with my results with this preparation in Staphylococcus infection of the pharynx, otitis media, and tonsillitis, I am suggesting it as an addition to our therapeutic armamentarium. It is available in sustained release form which provides the simplicity of twice-a-day administration since blood levels after 12 hours are still at the level of 8 to 10 mg. %. Its acetylation in the blood and urine are lower than that of any other sulfonamide, and crystalluria has not been reported. There have been no instances of blood dyscrasias.

The advent of the antibiotics caused a long eclipse in the use of chemotherapy, including the sulfonamides. You[22] called attention to this lapse of interest brought about by the dramatic results in the therapeutic use of penicillin and other antibiotics. Now the pendulum is swinging. Because of varying degrees of sensitization to penicillin up to actual fatal anaphylaxis, and because of the development of resistant strains of bacteria, resulting at times in superimposed infections during therapy as well as the occurrence of other undesirable side-

* Trade name Sul-Spansion (Smith, Kline & French Laboratories, Philadelphia).

effects, we are beginning to turn again to the sulfonamides, especially in those infections which can be safely and effectively controlled by them.

Furfural derivatives represent another class of drugs worthy of serious consideration in the treatment of these resistant staphylococcic infections. Probably nitrofurantoin is the most valuable member of this family, and may be used either by itself or to supplement one of the other forms of therapy.

Summary

Infections produced by the hemolytic Staphylococcus aureus present a variety of clinical and pathological forms ranging from mild pustules to fatal septicemia. Strains of this pathogen are steadily becoming more antibiotic-resistant. Laboratory diagnosis with information regarding strain sensitivity to various antibiotics is of prime importance. Droplet infections in nurseries can cause epidemics. Hospitals should maintain scrupulous cleanliness and observation of personnel, especially in the areas of pediatrics and surgery, where antibiotics are used more frequently. A tendency is developing to treat infections of less import and those occasioned by antibiotic-resistant strains with sulfonamides and other chemotherapeutic agents.

In severe infections, and in cases showing some degree of sensitivity to one or more of the antibiotics, combined therapy with two or more antibiotics often coupled with one of the synthetic chemotherapeutic agents is being employed more and more frequently.

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The Management of Sinusitis in an Allergic Individual

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About two decades ago occasional articles began to appear in the literature regarding the role of allergy in otolaryngology. In the last few years, however, the literature has been replete with papers by many writers who (1) stress the importance of allergy in this field, and (2) are more or less in agreement that allergy is an etiologic factor in an ever-increasing percentage of patients with ear, nose, and throat pathology.

Hansel,[1] in 1942, stated: "In an analysis of 455 patients referred to the Pediatric Allergy Clinic of Washington University we found 341, or 75%, had a definite respiratory allergy."

Shambaugh,[2] in 1945, stated: "At least 70 percent of chronic sinus infections and at least 90 percent of chronic nasal infections can be shown to have an underlying allergic factor responsible for the chronicity."

Ashley,[3] in 1949, stated: "The importance of allergy as an etiologic factor in pathology of the nose, throat and ear is still not universally recognized. As a matter of fact, a careful routine allergic history and an examination of the nasal mucous for eosinophiles will frequently show that a high percentage of all nasal symptoms are due to allergy and not to infection, and that even when infection is a factor, an allergic mucous membrane very often is the receptive media upon which the secondary infection becomes superimposed."

Gill-Carey,[4] in 1950, stated: "Only 30 percent of sinus disease is infective and 70 percent is due to some obscure pathological state which may be endocrine, allergic or dietary in origin."

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Hansel,[5] in 1956, stated: "80 percent or more of the problems encountered in the private practice of otolaryngology requires medical treatment."

If the foregoing statements are true, then one must conclude that infective sinusitis per se is not the common disease it is believed to be, but is rather a comparatively rare entity.

The treatment of sinusitis in general has undergone remarkable changes during the past three decades. When I was a senior house officer at the Massachusetts Eye and Ear Infirmary some 30 years ago, some form of radical sinus surgery was almost a daily procedure. Today, this type of treatment for sinusitis, even the simple Caldwell-Luc operation, is rarely performed.

This extreme reversal of treatment, in my opinion, is due to two major factors: (1) the better understanding of the normal physiology of the nose and sinuses resulting in a greater respect for same, and (2) the gradual recognition that allergy is a very frequent cause of nasal and sinus symptoms.

Putney,[6] Bryan,[7] and others have felt that the use of antibiotics has played a major role in the reduction of sinus surgery. However, in my opinion, the use of these agents has been of inestimable value in sinusitis with serious complications, such as orbital abscess, osteomyelitis, cavernous sinus thrombosis, meningitis, etc., but has played a very minor role in reducing the frequency of radical sinus surgery. The majority of nasal and sinus infections are rarely so serious as to indicate the use of antibiotics.

The purpose of this paper is (1) to re-emphasize the importance of suspecting the presence of allergy in all cases of sinusitis, and (2) to discuss the management of sinusitis in an allergic individual.

Before proceeding any further I should like to state that in every discussion it is of fundamental importance that all persons give the same meaning to the same words; otherwise misunderstandings frequently arise when different meanings are given to the same words. It is therefore of greatest importance to carefully define all key words about whose meaning there might be a difference of opinion. In this dissertation, the key words are (1) sinusitis and (2) allergic individual.

Sinusitis is defined in medical dictionaries and textbooks on pathology as an inflammation of the lining membrane of one or more nasal accessory sinuses. The word inflammation is further defined as a morbid change or a series of reactions produced in the tissues by an irritant. Many different types of inflammation are listed, but I should like to divide them into two main groups, allergic and infective.

My definition of an allergic individual is one who is so sensitive to various materials that exposure to small amounts of such materials will produce in such a person allergic or unusual tissue reactions, but will be insufficient to cause any such reactions in a normal individual. Such materials may consist of extrinsic factors, e. g., pollens, grasses, foods, cosmetics, etc., or intrinsic factors, such as emotional and psychic problems, autonomic nervous system imbalances, etc., that can and frequently do result in endocrine dysfunction.

Davison[8] and others do not classify autonomic dysfunctions as an allergy. I do not know the scientific answer to the mechanism of the true allergic response, but I am convinced that any emotional or psychic problem can produce a stimulus along the autonomic nervous system sufficient to cause an imbalance in the normal function of the various endocrine glands, such as the thyroid, pituitary, adrenal, ovarian, etc., and produce vasomotor responses similar in every clinical respect to those caused by any extrinsic factor, such as foods, pollens, dust, etc.

Jordan[9] stated: "I do not believe that all allergy is an antigen-antibody reaction by any means, and I include all autonomic dysfunctions, psychic changes, endocrine dysfunctions, etc., under the broad term, allergy. I don't believe allergy is a nose or an ear; it is a complete individual and that individual may show reaction to what we call an allergic nature to many stresses and strains."

Walsh,[10] in 1955, stated that he was unable to identify allergic factors in a large percentage of his patients with vasomotor rhinitis, but that endocrine, dietary, and psychosomatic disturbances play a major role in the etiology.

Selye,[11] in 1950, has shown that there are normal, physiological, and biochemical reactions of the body to shock, stress, and fatigue and that they cause depletion, or excessive liberation of the hormones of the adrenal cortex, with profound changes in various body structures.

Forman[12] sums up the definition very well by stating "Allergy is the sum total of unfavorable stresses and strains which the environment places upon the individual."

To summarize, then, sinusitis in an allergic individual means a morbid change or a series of reactions that take place in the lining membrane of the sinuses in a sensitive person who reacts abnormally when exposed to various external or internal stimuli in amounts too small to cause similar reactions in a normal individual.

Now that these terms have been clearly defined, let us proceed with the topic of this thesis, namely, the management of sinusitis in an allergic individual.

In order to most effectively manage sinusitis in such an individual, it is fundamentally important first to establish a correct diagnosis as to the type of sinusitis we are dealing with. To simplify the discussion I should like to classify the various types as follows:

I. Clear-cut allergic sinusitis
II. Clear-cut allergic sinusitis with nasal polypi
III. True infective sinusitis
IV. Infective sinusitis with nasal polypi

Types I and II. Allergic Sinusitis

Types I and II are similar in every respect except for the presence of nasal polypi in the second type; therefore, a discussion of the management of these two types will be combined. These two groups, in my experience, constitute the majority of all sinus cases. I much prefer to speak of these types as pseudosinusitis, for these patients complain of various sinus symptoms, but are suffering from frank allergic reactions rather than from infection of the sinuses. These people are frequently told by their physicians that they have "sinus trouble," which, to the average lay person, means pus or infection in the sinuses. But a proper and thorough rhinological examination, including x-ray study of the sinuses, only fails to substantiate any demonstrable sinus infection. Yet, these patients are too often subjected to persistent treatments with various nasal medications, antibiotics, and even repeated surgical procedures.

Unfortunately the statement made by Ashley[3] in 1949 still holds true today, almost 10 years later: "The general belief still persists among rhinologists that the great preponderance of nasal symptoms are secondary to infection of the sinuses or of the nasal mucosa itself, and that either surgery or persistent treatment with nasal decongestants, the sulfonamides or penicillin will cure most of these conditions. The failure to obtain results is still credited to poor or incomplete surgery, to insufficient local treatment, or inadequate treatment with the antibiotics."

Such procedures not only prolong, but very often increase the severity of the pseudosinus symptoms. The allergic congestive reaction of the mucosa is completely reversible, and in most cases the mucosa will be restored to normal by merely discontinuing the nasal treatments.

Since Types I and II are not infective but are frank allergic reactions of the nasal and sinus mucosa, the management should consist of the following: 1. A careful history should be taken, which should include a detailed family and past history and the history of any preexisting allergy; also a careful analysis of the home environment, occupation, social habits, cosmetics, medications and foods in relation to their symptoms. 2. A proper otorhinological examination should be made, including roentgen studies of the sinus cavities. 3. The presence of infection in the sinuses having been ruled out, all local therapy such as nose drops, irrigations, etc., and antibiotic treatments should be definitely prohibited. 4. This last procedure very often is sufficient to allow the allergic mucosa to subside to such a degree that many nasal polypi will almost miraculously disappear. However, if some polypi that have become fibrous persist and obstruct the nasal and sinus passages, a polypectomy will be indicated. 5. Occasionally it may be necessary to perform a submucous resection in order to correct an obstructive septal deviation or crush and infract enlarged cystic turbinates. 6. Finally, a very thorough search should be made for the allergic offender, no matter how obscure it may appear, and proper allergic treatment instituted as soon as possible, and a complete general physical examination should be performed on all these patients, with special emphasis placed on the endocrine system.

Type III. True Infective Sinusitis

This is usually an acute condition and I believe constitutes the least common type found in allergic persons. When acute infection is present, pus is retained in the sinuses by the inflammatory edema of the mucosa obstructing the sinus ostia. The judicious use of proper vasoconstricting drugs will maintain adequate shrinkage of the swollen mucosa, allowing the sinus secretions to gradually drain into the nose. This is attained by first gently spraying the swollen mucosa with an aqueous solution of 0.5% cocaine hydrochloride. The nasal cavities, especially along the floors, are then cleared of any thick secretions by means of a thin,

long, straight suction tube made specifically for this purpose. After this the middle and superior meatuses of the nose are gently swabbed with a fine cotton-tipped wire applicator moistened with 1% cocaine solution. Suction is again applied to the middle and superior meatuses to remove any secretions that might have drained from the sinuses. Thin cotton pledgets saturated and wrung out with the same solution are then gently placed into the meatuses and allowed to remain there for 20 to 30 minutes. On removal of these packs the sinuses are drained by spot suction, using a similar fine suction tube as described above, but having its tip bent at a 45-degree angle so as to be able to enter the meatuses. These treatments are supplemented by general supportive measures, such as bed rest, local applications of cold or heat, forcing fluids, and maintaining ample humidity of inspired air. This nasal procedure is repeated every two or three days, and usually three or four such treatments will result in complete drainage and cure of the sinus infection.

Antibiotics and chemotherapeutic agents are rarely prescribed unless the patient is quite toxic and has high fever or presents signs and symptoms of a possible serious complication. If the use of such agents is contemplated, a careful history is obtained as to which of these drugs the patient has had in the past and whether any reaction occurred, no matter how slight it may have been.

Hansel,[13] in 1954, stated: "It cannot be too strongly emphasized that the antibiotics should not be used indiscriminately. Most respiratory infections are mild and the use of antibiotics is not indicated. There are far too many occurrences of side effects which are worse or more serious than the original disease. There is evidence that the percentage is increasing and because of the failure of recognition of previous reactions, more serious and even fatal reactions are also on the increase. A positive history of allergy (active or inactive) in patients about to receive penicillin should receive some consideration as a higher incidence of the development of penicillin hypersensitivity should be expected."

Type IV. Infective Sinusitis with Nasal Polypi and Pus

Almost all of the sinus patients that fall into the category of Type IV represent long-standing chronicity of clear-cut allergic sinusitis with superimposed infection. Invariably this condition results from (1) failure to recognize the underlying allergic factors and properly treating them in time and (2) misuse of the antibiotics and the prolonged and pernicious use of local therapy such as frequent nasal irrigations, drops, and sprays.

In my experience, this latter fact is very often due to the malpractice of medicine by radio, television, and the "friendly pharmacist." I regret to state that frequently the local family physician is also at fault. Time and again patients have said to me, "The reason I have not visited a nose specialist sooner is because my doctor told me there isn't much that can be done for chronic sinus."

Although this condition may have persisted for months and even years, a goodly percentage of these patients obtain remarkably good results if the treatment as outlined for Type III is followed in conjunction with a thorough search for and removal of all allergic factors. This must include a thorough search for and elimination, if possible, of systemic conditions such as nutritional deficiencies, endocrine dysfunctions, and psychogenic factors.

In a considerable number of these patients the anticipated therapeutic response to the above measures is not obtained, since irreversible changes have taken place in the nasal and sinus mucosa resulting in hypertrophic, fibrotic polypi with obstruction.

If the infection is fairly well controlled, a short course of corticotropin (ACTH) or cortisone for four or five days may be prescribed. However, one must always bear in mind that these agents are easily capable

of producing very serious side-effects, especially if administration is prolonged. Therefore, extreme caution should always be observed when prescribing these drugs, and they should be prescribed only after the usual therapeutic measures have failed.

If after a fair trial of conservative local treatment, which should continue for several weeks, the infection and the obstructive polypi fail to respond, then some form of surgery is indicated.

These procedures should aim at correcting anatomic and pathologic obstructive mechanical conditions that are interfering with normal aeration and drainage of the nose and sinuses. Most of this can be accomplished by simple conservative nasal surgery such as a submucous resection, nasal polypectomy, and crushing and infraction of overgrown cystic middle turbinates. Occasionally, antrostomies and/or intranasal ethmoidectomies may also be necessary.

Hill,[14] in 1953, summed up this subject very well when he stated: "The days of ruthless sinus surgery of the nose and sinuses have past and in its place we have a wholesome regard for the integrity of the normal defenses and an understanding of the factors which make for chronic nasal symptoms such as allergy, endocrine disturbances and other constitutional conditions. To properly appraise all these elements requires careful history taking and an examination that embraces far more than the local inspection. If surgery is indicated it should be based on sound pathologic indications and should aim at adequate ventilation and drainage with minimum destruction of functioning tissues."

Extensive maxillary or frontoethmoidal sinus surgery is rarely indicated, and, if performed, should conserve the normal anatomical structure as much as possible.

General Comments

It has been repeatedly stated in the literature that the allergic individual is more prone to infections of the nose and sinuses. Judging from years of careful clinical observation of these patients, I cannot agree with this statement. As a matter of fact, it is my conviction that the true allergic individual is somehow unusually free from or resistant to true infectious colds, in spite of their self-made diagnoses of "frequent colds." Their "cold" symptoms are not due to the infectious common cold, which is the result of invasion of the nasal mucosa by some bacteria or viral organism, but are simply due to hyperactive normal physiological responses of the membranes to some external or internal exciting factor to which the patient is unusually sensitive. This is particularly true in persons whose allergic reaction usually manifests itself in the upper respiratory tract.

For many years I have felt that these normal hyperactive physiological responses cause some unknown biochemical changes, either in the general circulatory system or locally in the nasal secretions or mucosa, which definitely act as a defensive mechanism against the common cold viral invasion. Several questions present themselves to me, e. g.: 1. Is there some, yet undiscovered, defensive enzyme produced in the allergic nasal tissue? 2. Is there some biochemical agent formed that produces changes in the viscosity of the normal nasal mucous, as is evidenced by the typical thin mucoid or watery discharge from the allergic nose, that is bacteriocidal in nature? 3. Is there an alteration in the hyaluronic acid and hyaluronidase relationship which alters the permeability of the cell tissues? 4. Does the altered viscosity of the nasal mucous in allergic reactions so stimulate the activity of the ciliae as to make them a more powerful defensive factor? Proetz[15] and others have investigated the factors that control the normal activity of nasal cilia. A water medium is essential to normal ciliary activity. Proetz,[16] in 1933, pointed out that areas of dryness of the nose may lead to halting of the ciliary activity, resulting in the stasis of the overlying blanket of mucous. This, in turn, will allow any entrapped virus or bacteria to remain in contact with the nasal epithelium long enough to penetrate the

mucosa and cause infection. The typical allergic nasal secretions undoubtedly increase the ciliary activity, thus probably preventing the viral invasion of the mucosa.

Dixon, Hoerr, and McCall,[17] in their investigation of the nasal mucosa in the laryngectomized patients, stated: "It is believed that if the humidity of the room is kept high, the nasal mucosa moist, and the activity of the cilia normal, the danger of a patient contracting an upper respiratory infection is greately reduced."

Bronfenbrenner,[18] in 1940, was of the opinion that whenever antigen-antibody reactions occur in vivo, there is always a concomitant injury to the tissues of the host, as evidenced by both local and general symptoms. It appeared likely to him that the very mechanism which causes this injury to the host is also instrumental in bringing about such protective effects as the warding off of infection by immobilization of antigen by the antibody.

Allergy per se is not the etiologic factor in producing colds. However, if the allergy is allowed to continue untreated for long periods of time, it will cause irreversible changes in the nasal and sinus mucosa. These changes will prevent proper drainage of the nasal and sinus secretions, with eventual secondary infections. Thus allergy is not the actual cause but plays an important role as an underlying factor in sinus infections. It is for this reason that all allergic factors should be diligently sought for and treated as soon as possible in all cases of allergic sinusitis.

There is no question in my mind of the importance of thyroid dysfunction in relation to disease of the ear, nose, and throat. This interest, I believe, has been stimulated in recent years by reported results of studies on this subject by many investigators. Unfortunately, this interest is not equally shared by many general practitioners, pediatricians, and even internists. To this day, I frequently encounter difficulty in convincing a family physician of the importance of studying such a patient for possible hypometabolism. One must always bear in mind the distinction between hypothyroidism and hypometabolism. The latter refers to a more general endocrine disturbance, not necessarily confined to the thyroid. For this reason it is not wise to depend entirely on basal metabolism tests to determine whether the patient is in a state of hypometabolism. Although the basal metabolic rate is useful in diagnosing hypometabolism, it frequently is difficult to obtain a satisfactory test in these persons. Probably the best tests for thyroid function are the serum protein-bound iodine test and the uptake of radioactive iodine (I^{131}) by the gland, if one can be sure that the patient has not received iodides in any form previously. It is my conviction that hypometabolism is a very frequent cause of allergic sinusitis, and all cases should be studied from this standpoint and treated accordingly, if one expects a cure. Certainly no radical sinus surgery should be performed in these cases, unless complications are present, until hypometabolism has been ruled out or treated, if present.

Summary and Conclusions

Sinusitis in the allergic individual is defined and the various types found in such individuals are classified.

Emphasis is placed on the importance of differentiating between true clear-cut allergic sinusitis and infective sinusitis.

In the former type the management is to (1) prohibit any further use of local nasal sprays and irrigations, and (2) find the allergic offender, whether it be dust, pollen, food, cosmetics, or emotional or glandular disturbance, and institute the proper treatment as soon as possible. A cure is usually assured, allowing, of course, some time for the tissues to heal.

The latter type, allergic sinusitis with superimposed secondary infection, may be acute or chronic. The majority of cases are of long-standing chronicity and usually associated with many nasal polypi and much pus. Acute symptoms are conspicuous by their absence.

The management of the chronic type should consist of prohibiting any further use of local medications by the patient, draining the existing infection by local shrinkage and spot suction, together with simultaneous control of all allergic and constitutional factors. In a high percentage of cases this procedure will effect very satisfactory results. In some it may also be necessary to resort to conservative intranasal surgery, while the severer, resistant, complicated cases may be helped by the cautious use of cortisone and antibiotics. Rarely should destructive radical sinus surgery be performed. If such surgery is contemplated, then only such procedures sufficient to conserve, as much as possible, the normal anatomical structure and physiologic function should be performed.

Sinusitis with pus and acute symptoms, not associated with chronic obstructive nasal polypi, is not very common in the allergic individual. This acute type should be managed by packing the middle and superior meatuses with thin cotton pledgets wrung out of 1% aqueous solution of cocaine hydrochloride, followed by spot suction. This procedure is repeated every two or three days and supplemented by general constitutional treatment such as bed rest, control of pain, proper diet, etc. With this management a complete cure will result, in almost all cases, in a relatively short period of time. Occasionally it may be necessary to irrigate the sinuses after the acute symptoms subside. Antibiotic and chemotherapeutic agents should not be used routinely but should be reserved for those patients who are generally quite toxic or present any signs or symptoms of impending serious complications. The management of infections in allergic individuals should not vary from that in nonallergic individuals except for the added measures required to control the allergic factor.

The most important principle in the management of sinusitis in an allergic individual is to constantly think of allergy and to recognize it, no matter how obscure it may appear.

Finally, it is suggested that the true allergic individual is, for some unknown reason, unusually resistant to the invasion of the common cold organisms. It is also suggested that the reason for this may lie in the possible biochemical changes that probably take place in the local nasal tissue cells or secretions whenever endocrine dysfunctions are present. It would seem that this biochemical change is an attempt by the body to protect the slow functioning, hypometabolic, allergic individual from too frequent invasion by the highly contagious common cold organisms. Future scientific research of these biochemical changes that take place in relation to endocrine dysfunctions may hold the solution to the exasperating problem of "how to prevent the common cold."

416 Marlborough St. (15).

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A Clinical Report of Footplate Fenestration Published Sixty Years Ago

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While we were preparing a report to be presented before the Société belge d'Oto-Rhino-Laryngologie, the following observation by H. A. Alderton came to our notice (Hicguet and Van Eyck, 1947). The title of the clinical paper was "Trephining of Stapedial Footplate for Otitis Media Sclerosa," and it appeared in 1898. In view of the present extensive interest in stapes mobilization and footplate fenestration in otology, it seems of interest to call the attention of our colleagues to that old contribution, which, in view of its historical interest, seems to deserve publication *in extenso*.

So wrote Alderton:

Miss Lillian F. came under the writer's care Dec. 10, 1896, complaining of hardness of hearing and tinnitus in both ears, more marked in the right. The whisper was heard in the right ear at 4½ feet for the most audible sentences. Eustachian tube free.

Tuning fork reaction.

		Rinné	BC	BC	BC	B	=	=
Schwabach	AC	0	12	9	17	14	17
	BC	12	17½	8½	27	13	14
	T. F.	C-1	C	C1	C3	C2	C4

After treatment with the catheter, etc., for a number of weeks without improvement, recommended an exploratory myringotomy, followed by removal of the ossicles if any improvement was evidenced.

In March, 1897, the writer turned down the posterior-superior quadrant of the drum membrane as a flap, with the base on the level with the umbo, under cocaine anesthesia. Slight improvement in hearing. At once proceeded to cut the stapedius tendon, disarticulate the incudostapedial joint and remove the incus through the perforation made in the drum membrane. Neither the malleus nor stapes were disturbed.

No further improvement in hearing followed this operation; the drum membrane reformed perfectly without any reaction and the tuning fork tests following healing showed no material differences from those given above.

The patient was much disappointed in the result, notwithstanding the prognosis had been extremely guarded. As her livelihood depended on her hearing she begged that something else might be done, and, accordingly, in the fall of 1897, the writer again turned down the flap in the superior-posterior quadrant and undertook to remove the stapes, under cocaine. Upon traction being brought to bear on the crura with the hook these came away, leaving the footplate behind. Immediately the patient became very dizzy and had to be supported. After the dizziness had subsided sufficiently, she exclaimed that she heard better. Testing showed that she heard the whipser the length of the operating room, about 18 feet. The next day the hearing had diminished so that the whisper was only heard at 9 feet and in the course of the next few days the hearing distance went back to where it was before operation.

There was no inflammatory reaction and the drum membrane reformed perfectly.

Evidently the force exerted had been sufficient to break up the adhesions around the footplate and temporarily mobilize the stapes, but union having again occurred, the hearing returned to its former condition.

Having once heard, the patient would not believe there was not a chance that she might hear again, and perhaps, permanently. She finally induced the writer to try to trephine the footplate of the stapes.

On January 25th, 1898, with the help of Dr. Braislin of Brooklyn and Dr. Morrow of Albany, the writer again turned down the posterior-superior quadrant of the drum membrane, the malleus being still in its normal position, and with a guarded trephine drilled a hole through the

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Chargé du Département d'Oto-Rhino-Laryngologie de l'Hôpital Brugmann, Université de Bruxelles.

	Rinné	BC	BC	BC	BC	=	AC
Schwabach	AC	0	14	7	15	14	14
	BC	13	15	10	33	16	12
	T. F.	C^{-1}	C	C^{1}	C^{2}	C^{2}	C^{1}

stapedial footplate. Labyrinthine fluid escaped. No dizziness followed. The patient complained of marked autophony and bubbling noises in the ear. Also a strong commotion, "as if an old, out of tune, unstrung musical instrument had by some sudden shock been set into a quiver of unpleasant vibrations"; any vocal efforts made by others or herself, "seemed to be with the tremulo stop out." "Several times during the day there was a quick report, like the sudden breaking of a violin string."

In the next few days she had an occasional sensation of lightheadedness.

Her family physician thought there was a slight improvement in the hearing.

Feb. 19, 1898. Opening in membrane still present but no discharge, fenestra rotunda still in sight but the fenestra ovalis hidden by cicatricial tissue under posterior fold. Watch heard 3½ inches, whisper 3 feet, speech 3 feet. Weber slightly lateralized. Galton whistle 1,9. Tinnitus, hearing and everything else just the same as before the operation.

Tuning fork reaction.

March 5, 1898. Difficulty in closing opening made in membrane. Flap had sloughed off. No inflammatory reaction. Posterior border, base and anterior border of perforation show no growth, but the perforation is gradually closing down from the apex under the posterior fold. Hearing same as February 19th.

Conclusion

Apparently in otitis media sclerosa nothing permanent is gained by the attempt to remove or break through footplate of the stapes, and though nothing disastrous is to be looked for as the result of such an attempt, yet the writer, as the result of his experience, would counsel against the operation.

Note—September 7, 1898. Since the operation performed January 25th last, I have noticed some changes for the better in general hearing. I am not making the same amount of effort, every nerve strained to hear, and I can hear at longer distances and in larger places. Voices are clearer and sound more natural. My head feels more comfortable and clearer, not the "shut up" sensation then has been so long. This is particularly noticeable to me when lying on my left side. There is not the feeling of pressure on the right ear, nor of being completely shut off from everything external, noises in the head grow less, though never entirely gone. There is no very marked improvement of hearing in the right ear alone, but slowly, gradually, yet distinctly, there is improvement in general.

LILLIAN F.

Test at office, Sept. 8, 1898. Whispers heard a. d. 2 feet; a. s. 16 feet; both ears open, 16 feet. Conversational speech heard a. d. 2 feet; a. s. 22 feet; both ears open, 24 feet. Perforation nearly closed.

The hearing in the right ear is not improved, while the hearing in the left unoperated ear has been.

This perfectly well-detailed old observation given by Alderton shows that nothing truly new has been done recently by stapes surgery—except good results!

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Rhinoplasty. Past and Present

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Owing to modification of the surgeon's views and his enlarging experience, rhinoplasty, like any applied art, is subject to change. The rhinologist of the past, as a result of long trial-and-error experience, learned empirically the conditions for a successful rhinoplasty, but not the underlying principle. Thus he was unable to cope with unexpected perplexities, such as postoperative drooping of the tip, broadening, pinched "surgical" appearance, asymmetries, and cicatricial distortions. Without the proper concept, correction was attempted by this or that maneuver. The countless procedures recommended [1] added to the confusion of the novice and did not help the experienced operator, who had developed through the years a personalized method and was not likely to accept a suggested innovation in which he could not personally foretell the probable outcome.

The following remarks are addressed to the beginner, in an attempt to replace past complicated techniques with present simpler procedures.

Shortening of Nose

In the past shortening of the nose contemplated the removal of a triangle from the septum and upper lateral cartilages, to create a space into which the lower segment could be tilted.[2] While the procedure was effective, it was associated with many pitfalls. It required experience through trial and error, to determine the appropriate size of the triangle. If too much of the septum was removed, the result was a retracted columella (Fig. 1); if too little, a hanging septum. Furthermore, in cases where abnormal nasal width was due to hypertrophy or downward dislocation of the lower lateral cartilages, the removal of even the

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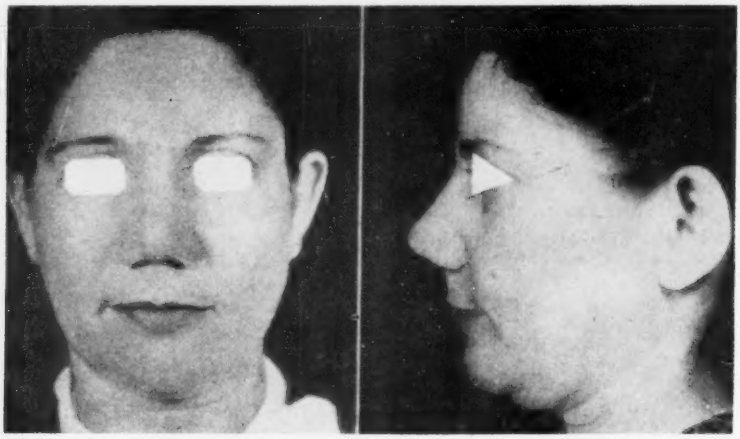
Fig. 1.—Retracted columella, due to excessive removal of septal cartilage. (Reproduced with permission from Goldman, I. B.: *J. Internat. Coll. Surgeons* 13:285, 1950.)



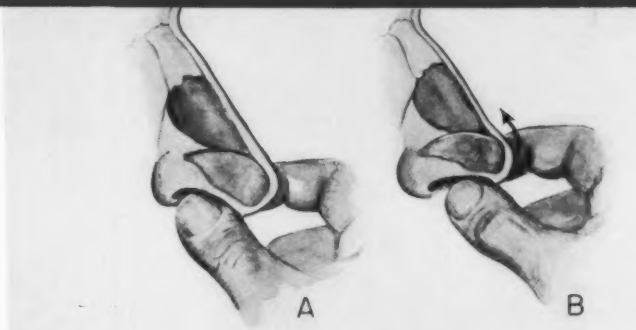


Figure 2

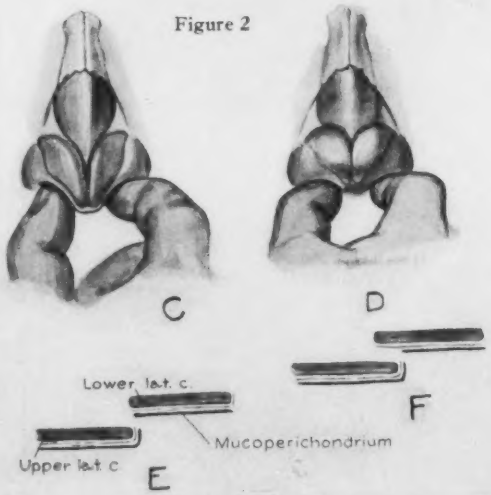


Fig. 2.—Shortening of nose by superimposition of lower cartilaginous vault over upper. *A*, columella grasped between thumb and forefinger. *B*, lower cartilaginous vault rotated over upper. *C* and *D*, serial frontal views. *E* and *F*, sagittal sections, showing relation of aponeurosis between upper and lower lateral cartilages. (from Fomon et al.: A.M.A. Arch. Otolaryng. 58:677, 1953.)

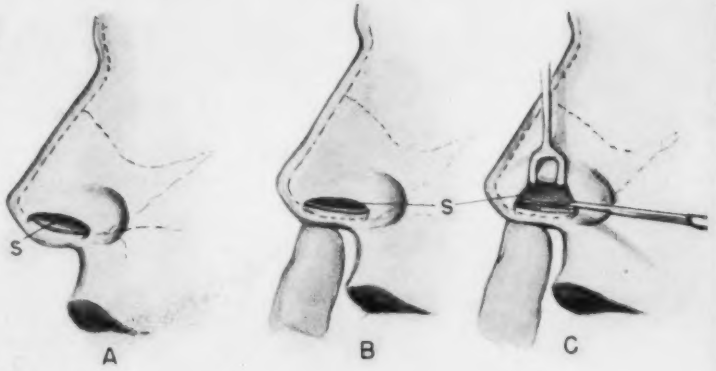


Figure 3

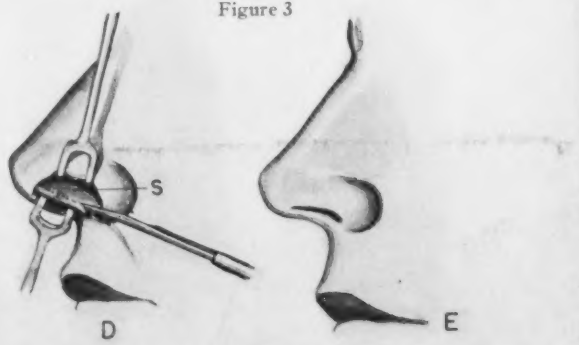


Fig. 3.—Method of estimating amount of septal cartilage to be removed. *A*, hanging columella, resulting from shortening of nose by overlap of lower over upper cartilaginous segment. *B*, columella raised along side of septum, to point 2-3 mm. below alar rim. *C*, ala retracted, and exact site on septum marked out. *D*, cartilage below marked-out site excised. *E*, result following excision. (from Fomon et al.: A.M.A. Arch. Otolaryng. 58:677, 1953.)

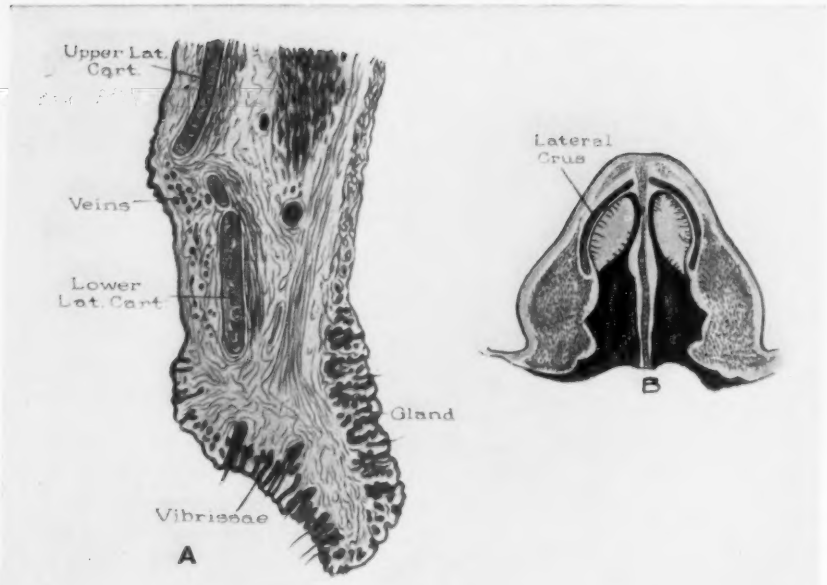


Figure 5

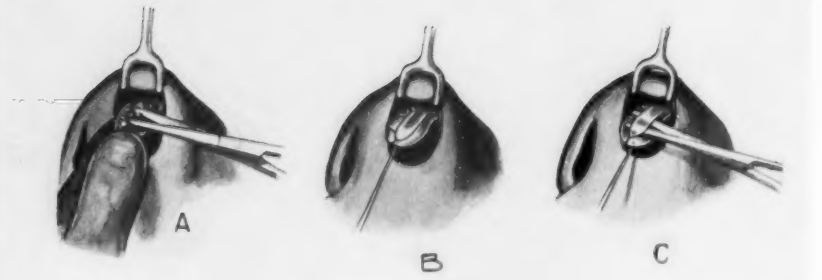


Figure 6

Fig. 5.—Sectional view showing relative size of cartilage as compared with total bulk of lobule. *A*, coronal section through ala. *B*, cross section. (from Fomon et al.: A.M.A. Arch. Otolaryng. 54:467, 1951.)

Fig. 6.—Removal of spring of lower lateral cartilages. *A*, soft tissue over cartilage removed. *B*, cross-cuts made through cartilage, to break spring. *C*, surface shaved. (from Fomon et al.: A.M.A. Arch. Otolaryng. 54:467, 1951.)

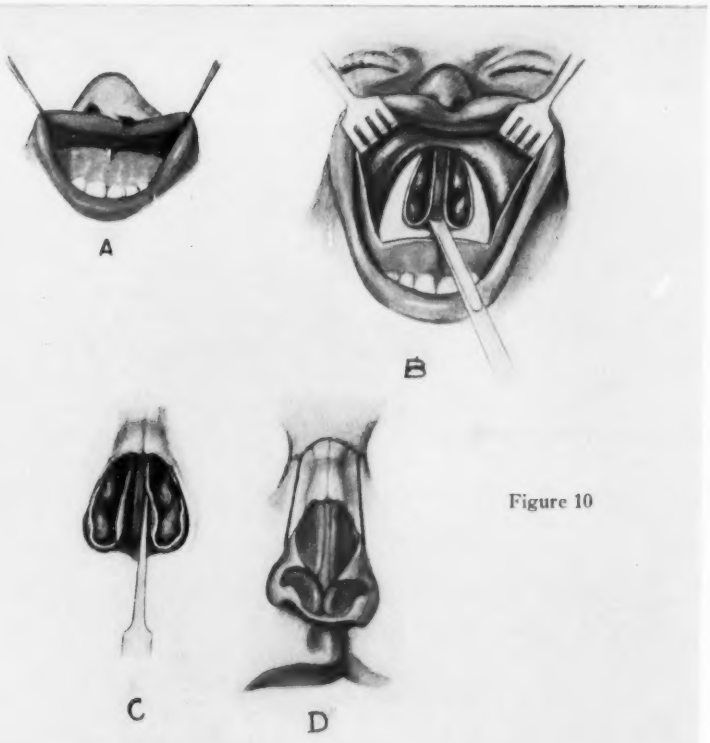


Figure 10

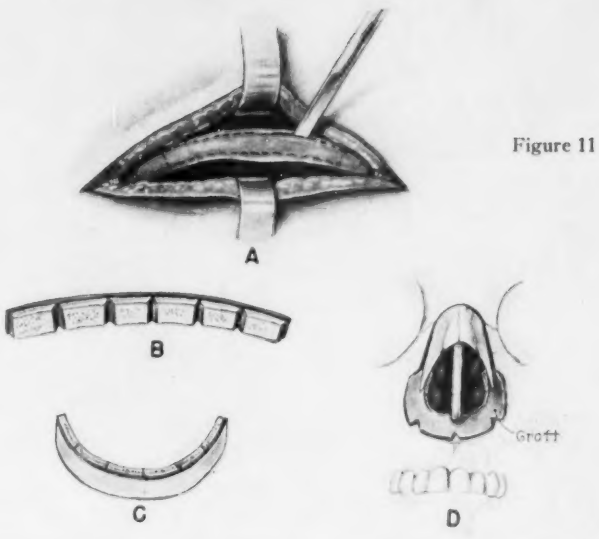


Figure 11

Fig. 10.—Correction of dish-face deformity. *A*, incision made along upper buccal sulcus, from molar to molar. *B*, soft tissue raised in plane above periosteum. Mucosa of nasal floor and septum elevated. Septum cut through along floor of nose. *C*, diagram, showing elevation of membranous floor of nose and septum. *D*, nasal pyramid mobilized. (from Fomon et al.: A.M.A. Arch. Otolaryng. 64:14, 1956.)

Fig. 11.—Procurement and modeling of graft. *A*, previously prepared pattern outlined on crest of ilium, and graft removed with chisel. *B* and *C*, graft modeled to pattern, and wedges excised from cancellous surface, to secure desired shape. *D*, graft placed over retruded maxilla and slid beneath previously mobilized pyramid for distance of 2-3 mm. (from Fomon et al.: A.M.A. Arch. Otolaryng. 64:14, 1956.)

smallest triangle would lead to a snubbing of the nose.[1] If an undue amount of the upper laterals was excised, a pinched "surgical" appearance ensued.[3] In addition, there was a tendency for the tip to droop. This was attributed to the stretch of the thin scar along the line of union between the upper and lower segments, the pull of the depressor septi nasi and the weight of the lobule. Various procedures [4-8] were employed to eliminate this sequela—such as overcorrection with silk or wire sutures, invagination of the septal cartilage into a columellar pocket, cutting of the depressor septi nasi, suturing of the lower to the upper laterals, and other methods too numerous to mention. None of these were consistently effective, as evidenced by the fact that 1 out of every 10 rhinoplasties, even in the best of hands, required revision for further shortening.

The present technique [9] contemplates removal of the triangle *after* the shortening, rather than before. This is accomplished by the rotation of the lower segment over the upper (Fig. 2), until the base of the nose forms an appropriate angle with the lip. This procedure converts the long-nose deformity into a hanging septum.

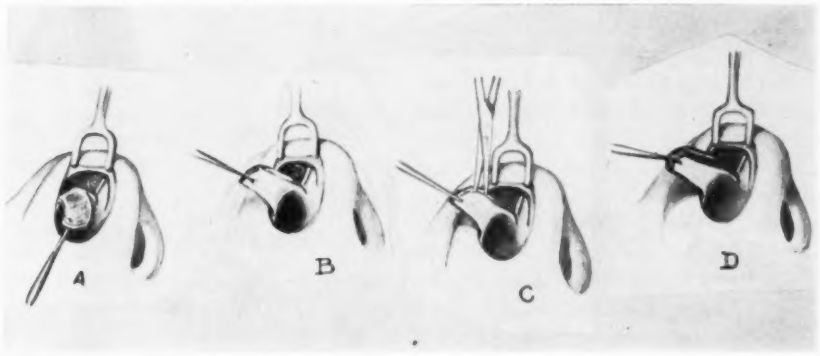
The columella is now raised on the side of the septum, to a point 2 to 3 mm. below the alar margin. The exact site on the septum is marked out and the portion below excised (Fig. 3). The upper lateral cartilages within the nasal fossa are left intact, unless they threaten to obstruct respiration.

This procedure removes many pitfalls for the beginner: (1) it accurately determines the size of the triangle to be removed from the septum; (2) it does away with the danger of a retracted columella and the pinched "surgical" appearance due to excessive removal of septal and upper lateral cartilages, and (3) it largely eliminates the postoperative drooping of the tip, by the broad adhesion resulting from the overlapping of the lower segment over the upper. In case of excessive length, the attachment is increased by enlargement of the raw area on the lower lateral cartilages by the separation of the vestibular skin without its removal (Fig. 4).

Shaping of Lobule

In the past, lobular deformities were assumed to be due to excess bulk of the cartilage, and shaping of the lobule was based on a sacrifice of the lower lateral cartilages.[2] For instance, projection was reduced by excisions from the crura; width was diminished by resections from the angles, and bulbousness by removal of a portion of the cephalic margins of the lower

Fig. 4.—Method of increasing attachment between upper and lower segment, to prevent postoperative drooping. *A*, lateral crus delivered, raw surface facing outward. *B*, lateral crus turned over, to bring vestibular surface into view. Note amount of raw surface for adhesion between upper and lower cartilaginous vaults. *C*, to secure broader adhesion, vestibular skin undermined with scissors but not removed. *D*, greater amount of raw surface for adhesion visible (from *J. Arkansas M. Soc.*)



lateral cartilages. Estimation of the amount to be taken away required a long experience, and the healing of the deskeletonized soft structures occasionally resulted in various asymmetries and distortions. Thus a lobule well modeled at the time of operation would not be so acceptable months later. A glance at Figure 5 will show that the cartilage in the lobule constitutes but a small part of the lobular mass, and that deformities here are due more to abnormal shape than to size.

The present technique envisages the reshaping of the cartilages by a removal of their elasticity through shaving or crosshatching (Fig. 6). The parts are then molded into the desired shape and splinted with adhesive, whereupon they heal to conform with the pattern of the splint. The technique has been described elsewhere.[11]

Narrowing of Nose

On cross section the nasal pyramid shows a series of gradually increasing equilateral triangles, the apices of which represent the nasal dorsum and the sides the lateral walls.[11] The base rests upon the maxillae. A change from this shape to that of an isosceles triangle, in which the length of the base appreciably exceeds that of the sides, constitutes the wide-nose deformity. Here a distinction must be made between a flat and a wide nose. For instance, on removal of a hump, a flat and not a wide nose results, since in a flat nose the lateral walls are equal in length to the base. All that is necessary here is to mobilize the walls and

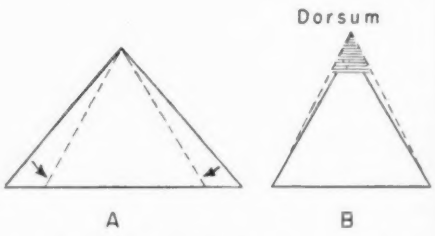


Fig. 7.—Narrowing of wide nose by transformation of isosceles into equilateral triangle. *A*, length of base decreased to equal that of sides by shifting lateral walls to midline. *B*, length of sides increased to equal that of base by use of modeled graft. (from Fomon et al.: A. M. A. Arch. Otolaryng. 59:200, 1954.)

to tilt them on a mucosal hinge to the midline, thus reconstituting the triangle.

The wide nose presents a different problem. It is narrowed by the transformation of the isosceles into an equilateral triangle by one of the following procedures:

1. Decreasing the length of the base (Fig. 7). The lateral walls are mobilized and shifted inward. Unfortunately, there is a tendency for a nose quite narrow at the completion of operation to return to its former width. This is explainable on anatomic grounds. Shifting the nasal walls inward creates a space between them and the maxillae. This space in time becomes filled with a callus, which on contraction is likely to pull the walls outward. We have tried in the past to solve this problem by the use of various splints, by suturing the upper cartilaginous vault, by through-and-through sutures tied over gauze rolls, and by internal wiring, but without much benefit. At the present time we are of the opin-

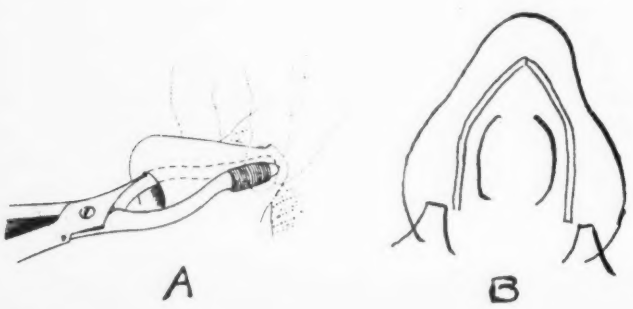


Fig. 8.—Prevention of postoperative broadening by shifting of frontal process beneath maxilla. *A*, Walsham forceps made to dislocate lateral wall beneath maxilla. *B*, diagrammatic representation. (from Fomon et al.: A. M. A. Arch. Otolaryng. 59:290, 1954.

ion that the most satisfactory procedure is to dislocate the lateral walls of the nasal pyramid beneath the maxilla (Fig. 8).

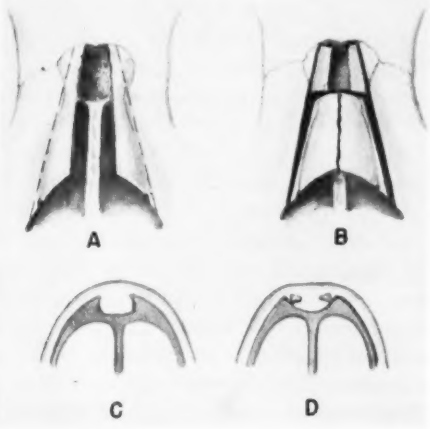
2. Increasing the length of the sides to equal that of the base. Following excessive hump removal, or in a Negroid nose, the lateral walls are too short, and the restoration of the equilateral triangle can be accomplished only by a lengthening of the sides with a graft (Fig. 7*B*).

Narrowing the root of the nose offers special problems. Here the nasal pyramid is a solid block of bone. Before the lateral walls can be shifted inward, it is necessary to chisel out a space. And since there is a limit to the depth to which this can be done with safety, and since the walls of the pyramid at the root are usually too short to meet above the chiseled space, satisfactory narrowing is not obtainable (Fig. 9). One will be forced either to cut the outer surfaces down to the required width or to equalize the lateral walls with a modeled graft. The latter procedure is more practical.

Hump Removal

Hump removal for the novice remains the greatest problem, for no landmark can at present be given as to the amount to be removed. The measurement of the nasal projection from the face (23 to 37 degrees) is a desirable objective from an artistic point of view, but of little surgical value. If one thinks of the nose on cross section as an equilateral triangle and of the hump as an isosceles triangle, in which the sides are longer than the base, it will facilitate the excision. By measuring the base and applying the measure to the sides, a more or less accurate idea of the amount to be removed can be obtained.

Fig. 9.—Diagram showing walls of pyramid at root too short to meet above chiseled space. *A*, chiseled space at root. *B*, walls incompetent to bridge space. *C* and *D*, diagrammatic representations. (From Fomon et al.: A. M. A. Arch. Otolaryng. 59:290, 1954.)



Selection of Patient

Factors that militate against a successful result are best avoided by careful selection of the rhinoplastic candidate. Failures are due largely to faults in architecture and to abnormalities in postoperative healing.

Architectural Defects That Militate Against a Successful Result

Harelip Nose.—In the adult harelip nose we are convinced that no operation limited to the nasal pyramid can remove the stigma of heredity.[12] A satisfactory result requires (1) the correction of the associated dish-face deformity; (2) repair of the nasal defect, and (3) reconstruction of the lip.

To overcome the dish-face deformity, the maxilla is built up with an osteoperiosteal graft. The approach for its introduction will depend upon the location of the defect. If around the piriform opening, the incision is made at the alarfacial junction. If it occupies the central part of the face, it is made at the gingivolabial sulcus (Fig. 10) and extended from molar to molar. The soft tissues are elevated in the plane above the periosteum, and the mucoperiosteum on the floor of the nose is raised, together with the septal membranes. The septum is cut through along the floor of the nose; an osteotomy is performed, and a properly shaped graft is introduced (Fig. 11).

To repair the nasal defect requires mobilization of all the elements and their

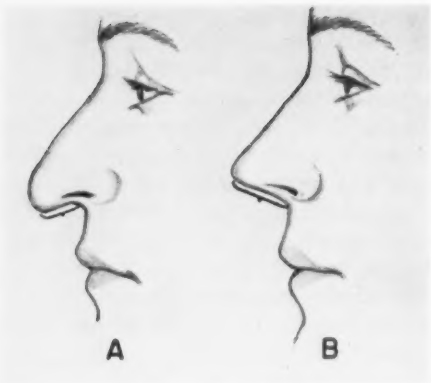


Fig. 12.—Increase of distance between apex and columellar-labial angle of basal triangle following shortening. *A*, distance between apex and septolabial angle before shortening. *B*, distance between apex and septolabial angle after shortening. (From Fomon et al.: A. M. A. Arch. Otolaryng. 64:456, 1956.)

reassembling according to rhinoplastic principles. The lip is then taken apart and united by one of the methods described in the literature.

Short Columella.[1]—In shortening the nose the distance between the apex (tip) and the columellar-labial angle is increased (Fig. 12). In the average nose sufficient "give" is present for the spanning of this distance. In the absence of such a "give," various procedures are employed to lengthen the columella, such as (1) gaining length at the expense of width; (2) adding tissue from the floor of the nose, or (3) camouflaging. Length may be simulated by a building up of the tip with subcutaneous buttons of cartilage. If these maneuvers do not suffice, and length can be obtained only by the transfer of tissue from a distance, cosmetic rhinoplasty is contraindicated.

Other architectural faults are abnormal relationship between the forehead and the nasal root (as in the Grecian nose with slanting forehead), depressed alae, seborrheic nasal skin, and unduly thick or thin subcutaneous tissue.

The condition of the septum is also a factor which determines the rhinoplastic result. Generally speaking, operative trauma detracts from the final appearance. Thus a rhinoplasty associated with a difficult submucous resection usually necessitates a revision. In the past we performed the rhinoplasty prior to the submucous resection, on the assumption that if the submucous resection were done first, the excision of the hump would remove the remaining dorsal buttress and result in saddling. With a better understanding of the architecture, we carried out the submucous resection prior to the rhinoplasty, on the grounds that it would facilitate the mobilization of the nasal pyramid. At present we do the two operations simultaneously, telling the patient that a secondary minor procedure may be required, to improve either the breathing or the cosmetic outcome.

Postoperative Faulty Healing

A rhinoplasty satisfactory on the operating table too often months later becomes unacceptable, owing to faulty healing. Until abnormal response of tissue to trauma is more thoroughly understood, faulty healing will hang like the sword of Damocles over all rhinoplastic surgery. The factors involved are age of the patient, capillary permeability and fragility, edema, ecchymosis, cicatricial distortion, and endocrine, hormone and enzyme dysfunction.

In relation to rhinoplasty the aging process begins about the fourth decade. In older persons the skin is less elastic and may not be able to readapt itself to the reconstructed nasal pyramid. It is likely to gravitate downward, forming a cutaneous hump. There is a natural predisposition for the piling up of connective tissue. The bones are more brittle and at operation subject to fragmentation. Calcification is impaired, with excess callus formation.

An increased permeability without capillary rupture will result in edema and petechiae. Fragility with vascular rupture leads to hemorrhage. A large number of tests based on petechial count following positive or negative skin pressure, together with the observation of bleeding and coagulation

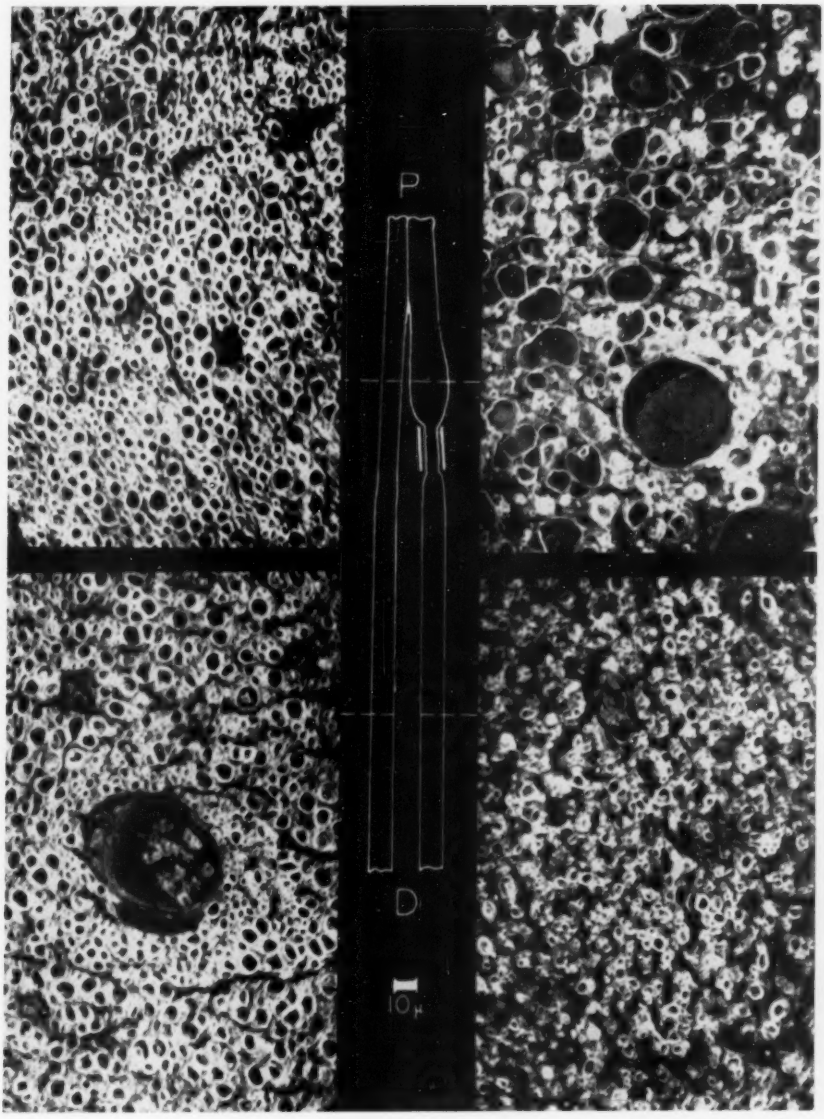


Fig. 13.—Effect of constriction on tissue. Unequal growth of constricted nerve fibers. Cross sections through a constricted nerve (right) and neighboring control nerve (left) at levels indicated in center diagram (mag. × 425). *P*, proximal; *D*, distal. The same nerve fibers, which are greatly shrunken distal to the constriction (lower right), have become blown up to giant dimensions just proximal to the "bottleneck" (top right); some fibers have become as wide as a small artery (compare the artery in the center of the distal control nerve at the lower left). (Reproduced from Northrop, J. H., and others: "Chemistry and Physiology of Growth," edited by A. K. Parpart, with permission of Paul Weiss and the Princeton University Press.)

time, we have found of no value. Since the cause lies in extravascular hydrostatic and osmotic pressures and in the nature of the capillary wall—including the adsorbed proteinaceous lining, endothelial cells, intercellular cement substance, capillary sheath, and ground substance—it is not likely that any one factor would be capable

of controlling capillary permeability and fragility. It is easy to make out a plausible case for therapeutic application, especially where there are no facts to counteract speculation. We have used without material benefit most of the suggested vitamins (P, C, and K) and hormones, singly and in combination. We have found that the best agent for the control of postoperative edema and petechiae is a pressure dressing applied to the face. In cases of bleeding, aside from packing, a blood transfusion is the most effective.

Hyperplasia of the collagenous fibers, producing a secondary dorsal convexity directly above the lobule, drooping of the nasal tip and unequal retraction of the alar rims, is attributed to tension of the scar formed at the union between the upper and lower segment following the shortening. Weiss[13] has shown that a constriction (scar) will result in a swelling proximal to the narrowing (Fig. 13). He believes that cytoplasm is constantly being produced in the cell and is in a continuous flow in a distal direction. If the movement is partially throttled by a constriction, the supply to the more distal regions is reduced, and the surplus piles up at the entrance to the narrows. If the constriction is removed, the effect is much like that of opening a flood gate. This is in conformity with clinical experience. Following the removal of the collagenous tissue, there is no recurrence of the hump. The above sequela may be prevented by an elimination of all postoperative tension, and this is accomplished by the overlapping technique.[9]

Other biologic factors that contraindicate a rhinoplasty are disturbances in the activities of the hormones, vitamins, and enzymes.

Summary

Past and present techniques of shortening, narrowing, and modeling of the lobule in rhinoplasty are reviewed. Observations are expressed on the selection of the patient, correction of the harelip nose, and short columella. Mention is made of biologic complications.

307 Second Ave. (3).

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Comments on a Fourth Year of Training for Certification in Otolarynogology

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Background

Just 10 years ago the American Board of Otolaryngology, after a review of the status of otolaryngology during and following World War II, decided the training of otolaryngologists needed strengthening. It ruled that all applicants for certification by the Board would be required to have three years of residency in otolaryngology if they began their training after July 1, 1950. At the time, there were not a few who expressed concern about this increase in the length of training. Today, however, there is no thought of a return to the former two years. There is universal acknowledgement that this move has been productive of great benefits to otolaryngology.

Ten years have passed since the announcement of this ruling. During these years, a more systematic and thorough system of appraising the residencies in otolaryngology in this country has been established. The appraisal body is known as the Residency Review Committee for Otolaryngology. It has succeeded the former Review Committee of the American Board, which, when it was in existence, passed judgment on residency training programs independent of the inspections of the American Medical Association and the Ameican College of Surgeons. When this system was in vogue, there was confusion and inefficiency, and the settlement of any differences between official inspections of the three bodies (and there were some) often took one to two years to iron out. The present Residency Review Committee, then, has three cooperative sponsoring organizations: the American Medical Association, the American College of Surgeons, and the American Board. There is equal representation on the committee, four otolaryngologists from each of the parent bodies. Each of the parent bodies has agreed to accept the judgment of these 12 men on any given residency. This means that approval or disapproval of a residency program, once it has been decided by the Residency Review Committee, has the simultaneous sanction of the American Medical Association, the American College of Surgeons, and the American Board. Order has come out of confusion. The Residency Review Committee is more efficient and its judgments are much more accurate and reliable. These judgments are the result of studied and often lengthy deliberations of 12 serious and dedicated men.

Over the past five years, the Residency Review Committee has processed an immense amount of data on otolaryngologic residency programs in the United States. The Committee meets twice a year, giving two days to each session. No other matters are considered save material pertaining to residency training programs in otolaryngology.

Good thinking and sound judgment have been used. As a result of these five years of deliberation, during which time the approval of more than a few residencies has been withdrawn (including, unfortunately, some university programs), a feeling has arisen amongst the members of this Committee that the 1948 action of the American Board of Otolaryngology, good as it was, was not enough. It has been felt that additional requirements were necessary to bolster the training of otolaryngologists. This feeling has not been crystallized by the Resi-

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dency Review Committee, for this is not its function. (Approximately 50% of the membership of the Residency Review Committee is made up of otolaryngologists who are not directors of the American Board of Otolaryngology.) A formalization into a regulation can only properly come from the American Board of Otolaryngology. Such action has been taken by the Board, but it should be noted that this action did not arise out of thin air, nor was it the result of pressures by individuals or groups outside of otolaryngology. It was as a result of the combined opinions of the men who are in the best position to know the strengths and the weaknesses of otolaryngologic residencies throughout the country, e. g., the members of the Residency Review Committee, and the men who see these same strengths and weaknesses manifest in the examination of candidates, e. g., the members of the Board.

The Action Taken

At a meeting of the American Board of Otolaryngology held in October, 1957, in Chicago, the Board voted to add an additional year of training to the three-year training period in otolaryngology. The Board made public announcement that the additional year was to be devoted to straight surgical training.

At a subsequent meeting held in San Francisco in May, 1958, the previous action was modified. At this meeting the American Board of Otolaryngology reaffirmed the requirement of a four-year approved residency training program, at least three years of which shall be in otolaryngology. The Board modified the requirement of one year of general surgery. The additional year of training may be devoted to otolaryngology or to other fields, such as surgery, medicine, pathology, basic sciences, or research.

Some Specific Reasons for the Action Taken

1. During the early part of this century some of the rightful functions of otolaryngology were lost by default. Occupied with infections of the sinuses and ears, the otolaryngologists of the country neglected to keep pace with advances made in the field of nasal and facial fractures, nasal deformities, and regional plastic surgery. Also, with a few notable exceptions, the major surgery for malignancies of the sinuses, the larynx, tongue, and in the neck was not included in the training of otolaryngologists. All of this requires excellent surgical technique and sound surgical judgement, in addition to a knowledge of the physiology and function of the structures involved, and an understanding of the general nutritional problems encountered.

2. In the past five years too many of the residency programs in otolaryngology have graduated men whose surgical record was deficient. (It is a requirement of the Residency Review Committee that a program under review must submit to the committee a list of the operations performed in that residency by the staff and by the residents.) The paucity of procedures in several programs has been notable. In a majority of such instances, otolaryngology has been but a division of the Department of Surgery, and these procedures were not available to the residents in otolaryngology. (About 60% of all the residency programs in otolaryngology are under control of the Department of Surgery in their institutions. In a goodly proportion of these, budgets are insufficient; an adequate number of hospital beds are not available, and little or no research is in progress.)

3. The tempo of residency training has slowed. Many residents are married, and the end of a day's work is often 5 o'clock, and not 11 or 12 o'clock at night, as it was in former days. There is now more to teach and less time in which to do it.

4. During the past 10 years there have been an increasing number of so-called oncologic surgeons trained. These surgeons are supposedly trained to handle surgically malignancies in any location in the body. In the knowledge of yesteryear, this new

specialist might be a boon to medicine, but in the light of modern medicine, with the necessity for knowing much about function, as well as anatomy, and with the availability of diagnostic methods and armamentaria which are best employed by specialists in their use, oncologic examination, surgery, and follow-up are not equal to that which can be performed by well-trained specialists in local areas. Otolaryngologic residents must have adequate training in this area.

5. Another important reason is the action taken by the National Institute of Health. A number of years ago this body felt that ophthalmology needed bolstering and spent some of its energies and monies for better training in ophthalmology, and the teaching of ophthalmology has improved. The National Institute of Health now feels that the teaching of otolaryngology needs strengthening; in fact, it is a specialty needing help more than most others. It is planning to spend thousands of dollars over the next 10 to 20 years for the training of teachers and research men in the field of otolaryngology. This is the opinion of a group outside of otolaryngology. This is significant.

6. The most important reason for an additional year of training in otolaryngology is the recent broadening of the field. Better training is needed so that future trainees will have a working knowledge of the many facets of modern otolaryngology. The scope of otolaryngologic surgery now includes procedures which are as complicated and demanding as any in the field of surgical endeavor. (Rehabilitation service for some of these patients demands a thorough knowledge of many techniques.) But there is also in this broad field the most delicate surgery of all—microscopic surgery of the ear. A very large portion of all allergy falls within the scope of otolaryngology. A modern otolaryngologist must know much about audiology. He cannot practice intelligently without this knowledge. Yet this science has grown to the point where studies at the doctorate level are required to encompass its broad reaches. A practical working knowledge of endocrinology is needed by the modern otolaryngologic specialist. Research in all these areas is expanding significantly. From what was thought to be a narrowing specialty in the mid-forties, otolaryngology has grown to become one of the broadest and most inclusive specialties of all. This is demonstrated when medical students are adequately exposed to it. Their response is usually one of amazement at the scope of the specialty, and the opinion they develop is at least one of respect, and often this is followed by an active interest.

How Will It Work?

The May, 1958, action of the Board of Otolaryngology purposely made the extra year of training flexible. Surgically oriented residents may spend one year of the four in straight surgery and yet receive credit for the year. Likewise, medically oriented residents may spend a year in medicine.

It may be asked, "Why should any surgery be included in the training of one who wishes to confine his practice to the medical aspects of otolaryngology?" The answer to this is that the very best internists are the ones who attend the surgery performed on their patients and know what is done, so they can intelligently treat them in the future. Medical otolaryngologists (they exist, and the good ones should be respected and admired) will be better specialists if they have had some surgical experience.

Various other combinations are approved. It is conceivable, for instance, that a resident may take two years of ear, nose, and throat training, spend a year in otolaryngologic research, and then take a fourth year of otolaryngology. He should make a most valuable senior resident.

If the choice is a year of surgery, this year should be oriented to the needs of the otolaryngologic resident. During the year of general surgery, the resident should receive training in patient care, which would include study of fluid and electrolyte balance, wound repair, shock, flaps, grafts, and dietary deficiencies of tube-fed patients. Operating room experience in the areas of

interest to the otolaryngologist, namely, neck surgery, chest surgery, neurological surgery, etc., should be included. In general, it would appear advisable to place this year of surgery between the first and third years of the otolaryngologic residency, which will be after the resident has some knowledge of what should be gleaned from this extra year. This surgical experience can be obtained in the same hospital in which the sponsoring otolaryngological department functions or in a neighboring institution if it appears better values can be realized. It is only necessary that a qualified surgeon be in charge of the approved surgical service which is to train the otolaryngologic resident.

Probably many of the otolaryngologic residencies of the country will make this program a straight four years in otolaryngology. Where the facilities of a department are such that the material, the teachers, and the armamentaria are available and of good standing, this will be the sensible arrangement.

A Look to the Future

The American Board of Otolaryngology has realized it must make moves which will strengthen the teaching of otolaryngology. With a long look into the future it has made the present ruling, i. e., an extra year of training. Otolaryngology has expanded. There is no denying this. Almost surely it will expand further (the bioacoustics of space travel, for instance). The extra year of training has been added to properly prepare residents for the very broad field now emerging. In the future not everyone will practice the entire field, but everyone should have training in all ramifications, so proper diagnoses, referrals, and follow-ups will be made. Only complete training will accomplish this.

Apparently, there is a need for more otolaryngologists in the United States today. One school of thought may argue for lower standards, so more practitioners will be produced. The time to increase standards is when there is a need for additional workers and when expansions in scope demand additional training. This is the situation today and is one of the reasons why the change has been made. It is believed that the response of future medical students will be adequate and rewarding.

This proposed year of additional training has seemed startling to some and to others unnecessary. This is probably so because not everyone is familiar with all this background material which has been available to the members of the Board. What everyone should remember is that this move has been made in view of the facts above outlined. It has been done in order to strengthen otolaryngologists and thus to strengthen otolaryngology itself. All other reasons are subservient to this.

1100 E. Genesee St. (10).

Primary Tumors of the Infratemporal and Pterygopalatine Fossae

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Neoplastic diseases which occur in the infratemporal and the pterygopalatine fossae are rare and unusual in their manifestations. During their early phase, tumors in this area may cause symptoms similar to well-known neuralgias. Frequently, the teeth will be extracted or the patient may receive treatment for sinusitis. A diagnosis of Costen's syndrome, Sluder's syndrome, or psychoneurosis may be made before the true nature of the disease is found. At the onset, these tumors do not extend externally beyond the confines of the fossae. Therefore, they do not present physical findings within the oral cavity, nose or nasopharynx. X-ray examination is usually normal until further growth has occurred.

Tumors which occur in the infratemporal fossa present symptoms that are distinctly different from those which arise in the pterygopalatine fossa or the sinus of Morgagni. In this respect, an interesting differential diagnosis can be made if the anatomy of this area is kept in mind. The progress of the neoplastic disease can be followed or predicted according to the adjacent anatomical areas which become involved.

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Anatomy [1]

The infratemporal (zygomatic) fossa (Fig. 1) is an irregularly shaped cavity,

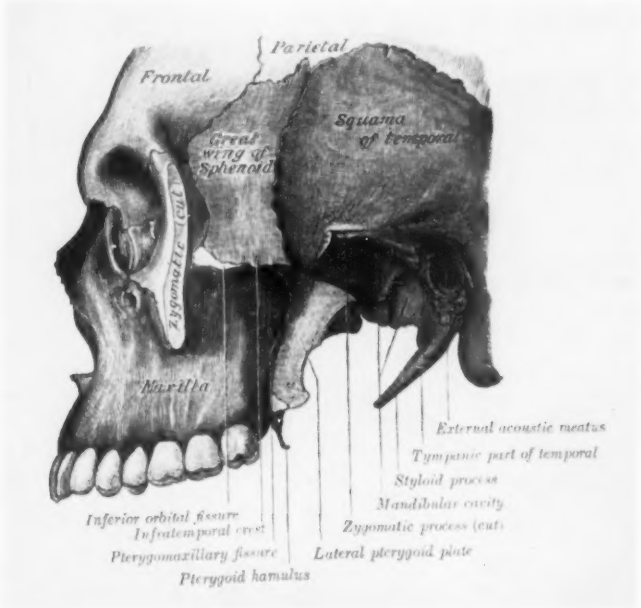


Fig. 1.—Left infratemporal fossa. (Reproduced with permission from Gray's Anatomy, Ed. 26, Lea & Febiger, New York.)

situated below and medial to the zygomatic arch. It is bounded anteriorly, by the infratemporal surface of the maxilla, and the ridge which descends from its zygomatic process; posteriorly, by the articular tubercle of the temporal and the spina angularis of the sphenoid; superiorly, by the great wing of the sphenoid below the infratemporal crest, and by the undersurface of the temporal squama; inferiorly, there is the alveolar border of the maxilla; medially, the lateral pterygoid plate. The contents of the infratemporal fossa consist of the lower part of the temporalis muscle, the pterygoidei internus and externus, the internal maxillary vessels, the mandibular nerve, and the maxillary nerve. The foramen ovale and the foramen spinosum open on the roof of the infratemporal fossa. The alveolar canals are on its anterior wall. At the upper and medial part of the fossa are two fissures, which meet at right angles, the horizontal limb being named the inferior orbital, and the vertical one, the pterygomaxillary. The inferior orbital fissure affords communication of the orbital, temporal, infratemporal, and pterygopalatine fossae. This fissure transmits the maxillary nerve, the infraorbital vessels, ascending branches of the sphenopalatine ganglion and veins. The pterygomaxillary fissure connects the infratemporal fossa with the pterygopalatine fossa, and transmits the internal maxillary artery and veins.

The pterygopalatine (sphenomaxillary, pterygomaxillary, sphenopalatine) fossa (Figs. 2 and 3) is a small triangular space at the junction of the inferior orbital and pterygomaxillary fissures, and is placed beneath the apex of the orbit. It is bounded superiorly, by the undersurface of the body of the sphenoid, and the orbital process of the palatine bone; anteriorly, by the infratemporal surface of the maxilla; posteriorly, by the base of the pterygoid process and the lower part of the anterior surface of the great wing of the sphenoid; medially, by the vertical part of the palatine bone with its orbital and sphenoidal processes.

The pterygopalatine fossa communicates with the orbit by way of the inferior orbital fissure, with the nasal cavity by means of the sphenopalatine foramen, and with the infratemporal fossa through the pterygomaxillary fissure. Five foramina open into this region. The three on the posterior wall are the foramen rotundum, the pterygoid canal, and the pharyngeal canal. On the

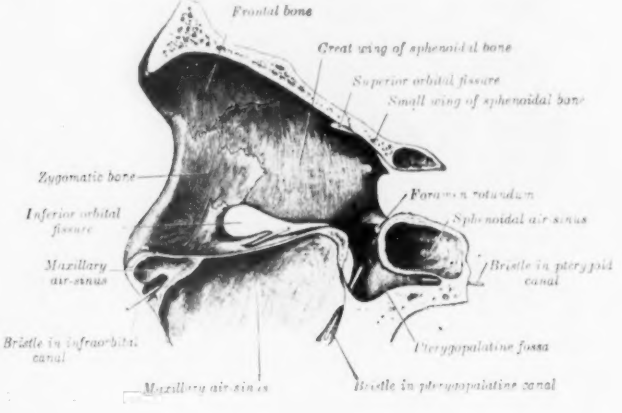


Fig. 2.—Lateral wall of the right orbit. (Reproduced with permission from Gray's Anatomy, Ed. 26, Lea & Febiger, New York.)

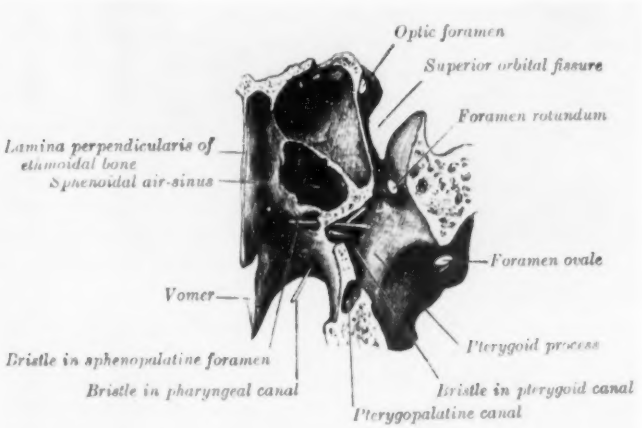


Fig. 3.—A section showing the posterior wall of the pterygopalatine fossa. (Reproduced with permission from Gray's Anatomy, Ed. 26, Lea & Febiger, New York.)

medial wall is located the sphenopalatine foramen, and below it lies the superior orifice of the pterygopalatine canal. The fossa contains the maxillary nerve, the sphenopalatine ganglion, and the terminal part of the internal maxillary artery.

The sinus of Morgagni is an important area in Trotter's syndrome. The superior constrictor muscle forms at its upper portion an inverted arch. This space between this upper border and the basilar process of the occipital bone is devoid of muscular fibers, but is closed by the pharyngeal aponeurosis. This gap, which is bridged over by the pharyngeal aponeurosis, forms the sinus of Morgagni and constitutes the lateral wall of the nasopharynx. From before backward,[2] the structures which are found within or adjacent to the sinus of Morgagni are the tensor veli palatini muscle, the Eustachian tube, and the levator veli palatini muscle. The fossa of Rosenmüller is posterior to the cushion of the Eustachian tube. The lower limit of the sinus of Morgagni is level with the floor of the nose. The anterior limit is about ¼ in. in front of the posterior margin of the septum.

The buccinator (buccal, long buccal) nerve is important in the symptomatology of the infratemporal fossa. The mandibular nerve gives off the anterior division which is composed of both sensory and motor fibers.[3] The buccinator nerve is the largest branch of the anterior division. It passes laterally between the two heads of the external pterygoid muscle and then downward and forward over the lower head of that muscle, under cover of the temporalis and masseter muscles, to reach the surface of the buccinator muscle near its lower border. In this area, it receives sensory stimuli from the skin and mucous membrane of the mouth.

Differential Diagnosis

The differential diagnosis would include several syndromes. Other related diseases which must be considered in the differential diagnosis involving this region are infection,[4,5] tumors of the parotid gland, mandible,[6-8] zygoma,[9,10] and maxilla.[11,12] They will present similar physical signs, but the difference will usually become apparent by further diagnostic procedures.

Infratemporal fossa tumors will most frequently cause symptoms of pain over the distribution of the mandibular nerve. The pain is constant and becomes increasingly worse with time. The buccinator nerve passes through this area and is likely to be interrupted by the lesion, causing paresthesia or anesthesia of the skin at the corner of the mouth and in the buccal mucous

membrane. Later, anesthesia of all branches of the mandibular nerve may occur. The tumor mass may present itself externally through the notch of the mandible or into the vestibule of the mouth in the area of the buccomaxillary fold. Trismus and deviation of the mandible will occur. As a later development, the Eustachian tube becomes obstructed, and there is fullness of the soft palate and nasopharynx.

Trotter's syndrome[13-15] was first described in 1911. Since then the syndrome has been reviewed by Gardham[16] in 1929, and more recently by Asherson[2] in 1951. Trotter's original paper described a triad: deafness, pain, and anesthesia in the area of distribution of the mandibular nerve, with subsequent asymmetry of the appearance of the palatal muscles. As the disease progresses, further involvement of the pterygoid muscles causes trismus, swelling below the zygoma, and finally metastasis to the neck. The tumor, an endothelioma, in Trotter's case did not encroach upon the lumen of the nasopharynx, but spread into the submucosa and deeply into the sinus of Morgagni.

The pterygopalatine fossa syndrome, with initial involvement of the maxillary nerve traversing the foramen rotundum, will cause pain or anesthesia throughout its distribution. Subsequent symptoms will more likely affect the orbit and its contents.

Jacod's syndrome[17] develops from a sarcoma of the Eustachian tube. Jacod described two cases in which its main direction of growth was toward the middle cranial fossa where the second, third, fourth, fifth, and sixth cranial nerves were affected. This syndrome produced a combined clinical picture of both Trotter's and the pterygopalatine fossa syndrome.

Costen's syndrome[18] is described as a malfunction of the temporomandibular joint, resulting in a symptom complex of pain in the region of the occiput, vertex, ear, and tongue. The ear feels congested. Frequently crepitation of the temporomandibular cartilages may be felt because of poor dental support in the molar region. X-ray evidence of temporomandibular joint pathology will aid in the diagnosis. Beyers[19] feels that Costen's syndrome should be considered in every case of recurring facial pain. A simple test consists of placing a cotton batten or rubber pad between the mandible and the maxilla, where proper occlusion of the molar teeth is lacking. The pain will be relieved by providing the molar occlusion.

Sluder's syndrome[20] was first described in 1918. Sluder placed all obscure aches and neuralgic pains of the lower half of the face into a group which he considered to be sphenopalatine neuralgia. In this condition the pain may radiate to the root of the nose, eye, zygoma, maxilla, upper teeth, ear, mandible, and lower teeth. It may also extend into the shoulder and arm.

Incidence

Primary tumors in these fossae are rare. Ehrlich[21] reported the successful removal of a mixed tumor from the pterygopalatine fossa. Asherson[2] and Trotter[13-15] felt that all pterygopalatine fossa tumors are metastatic in origin. In Trotter's syndrome, invasion of the infratemporal fossa may occur late in the disease. A search of the literature reveals that primary tumors of these fossae, with the presenting symptomatology and physical signs, have not been previously recorded.

The histology of the first case to be reported was that of a low-grade chondrosarcoma (Fig. 4). Chondrosarcoma constituted 7.6% of primary malignant tumors in the registry of bone sarcoma of the American College of Surgeons.[22] A total of 10 cases of chondrosarcoma of the maxilla since 1916 was reported by Paterson.[23] Single cases have been reported as occurring in the nose,[24,25] mandibular joint,[6] occipital bone,[26] sphenoid,[27] and coronoid process of the mandible.[7] Chondrosarcoma is found most commonly in the long bones. The most frequent age of occurrence is 40 years.[28]

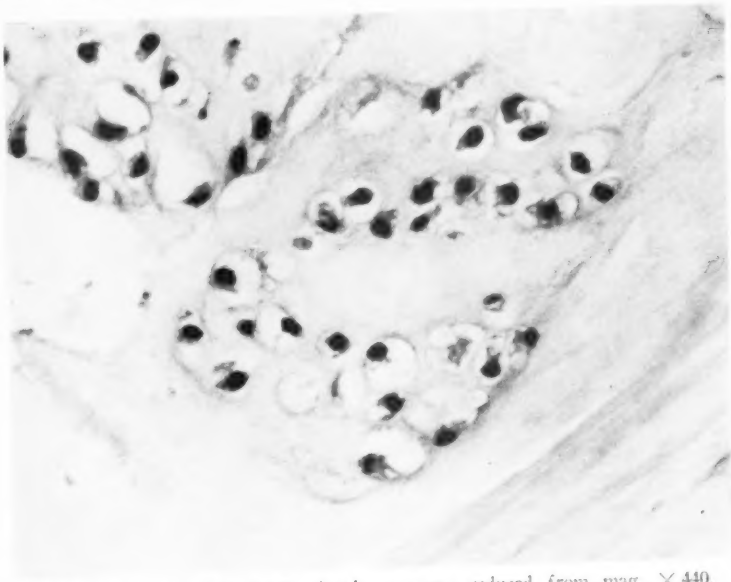


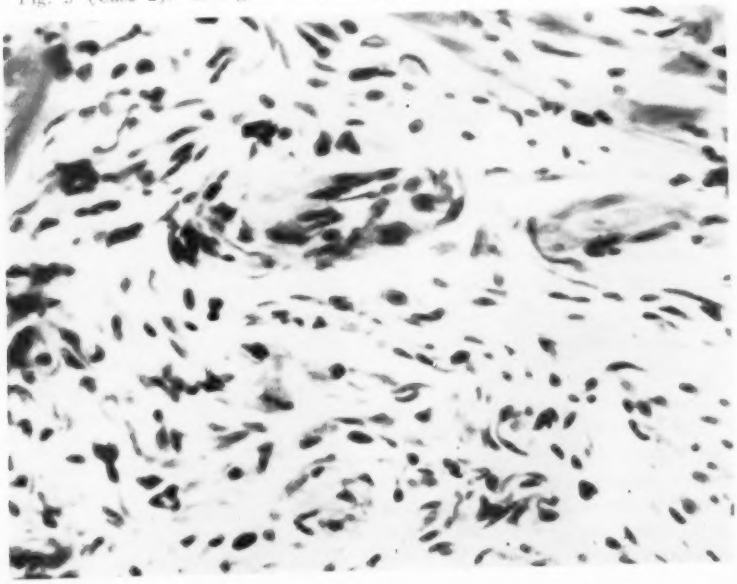
Fig. 4 (Case 1).—Low-grade chondrosarcoma; reduced from mag. × 440.

The second case to be reported was of neurofibrosarcoma (Fig. 5). This tumor, also known as malignant schwannoma, may occur at any age, but is commoner in the later years of life.[30] They are commoner in the mandible than in the maxilla.[31]

Embryology

Lichtenstein[28,29] believe that chondrosarcoma develops from full-fledged cartilage. The sphenoid, occipital, ethmoid, and nasal capsule are portions of the head that pass through a cartilaginous phase.[24] The bones

Fig. 5 (Case 2).—Low-grade neurofibrosarcoma; reduced from mag. × 440.



of the cranial vault, being ossified directly from primitive connective tissue, are not likely to develop primary cartilaginous tumors.[32] The maxillary sinuses appear about the fourth month of fetal life as an outgrowth of the lateral wall of the nasal sac. Cartilage is carried from the cartilaginous nasal capsule into the maxilla.[33] The mandible is derived from Meckel's cartilage. It has been shown that the coronoid process can have a cartilaginous origin distinct from that of Meckel's cartilage.[7] From an embryological standpoint, it is possible for chondrosarcoma to develop in the maxillary bone. A chondrosarcoma in this location, however, would be more likely to present itself in the buccomaxillary recess, in the vestibule of the mouth, or in the more visible areas of the maxilla. The portion of the infratemporal fossa most likely to be the site of origin in this first case reported would be the cartilage of the temporomandibular joint at its medial anterior surface, the great wing of the sphenoid, or the lateral pterygoid plate. In this location it could remain for a considerable period of time without causing symptoms. During gradual expansion, the external pterygoid muscle would become atrophic. The buccinator nerve which passes between the two heads of this muscle would become interrupted or compressed. The internal pterygoid muscle would offer resistance to easy spread in an inferior direction, thereby favoring a path of least resistance through the notch of the mandible.

In neurofibrosarcoma, the Schwann cell plays a predominant part in the growth of neurogenic sarcoma. The Schwann cells are embryologically from the neural crest. The tumors, although morphologically mesodermal in appearance, are ectodermal in origin.[31]

Report of Cases

Case 1.—A 40-year-old white man, who had previously been in good health, first consulted his local family physician and dentist in May, 1954, regarding a persistent pain over the left side of his face of several weeks' duration. X-rays of the teeth and the temporomandibular joint were normal except for a slight erosion of the left condyle of the mandible along its anterior border. This x-ray finding was believed to be due to a congenital abnormality. The pain became increasingly severe with radiation above the left ear. There was not any trismus or difficulty in chewing. Examination in November, 1954, revealed a slight asymmetry of the face which had not been noticed previously (Fig. 6). Palpation revealed a hard fixed mass below the left zygoma filling the region of the notch of the mandible. The mandible closed to the right of the midline with lesser horizontal mobility of the jaw to the right side as compared with the left. A small area of anesthesia was found at the corner of the mouth (Fig. 6). The remainder of the examination, including the nasopharynx, paranasal sinuses, and mouth, was normal. Complete blood count, urinalysis, and blood serology were normal. The pain was severe.

Fig. 6 (Case 1).—Primary tumor of the left infratemporal fossa presenting through the notch of the mandible. A small area of anesthesia at the corner of the mouth was caused by compression of the buccinator nerve as it passes through the infratemporal fossa.

In December, 1954, exploratory surgery was done under general anesthesia. The left parotid gland was exposed. The facial nerve was located at the stylomastoid foramen and followed anteriorly. The parotid gland over the masseter

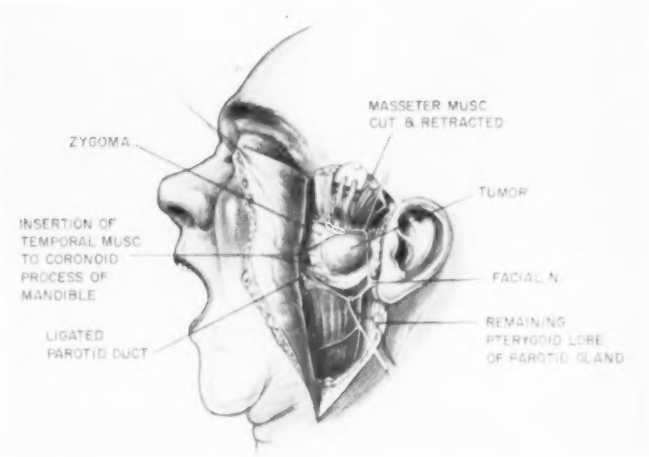


Fig. 7 (Case 1).—Exposure of the encapsulated chondrosarcoma after the parotid gland was removed and the masseter muscle divided and retracted.

muscle was removed. The muscle was divided in a transverse direction, and, upon retraction, a smooth encapsulated tumor mass was exposed. The tumor protruded from the notch of the mandible (Fig. 7). Medially, it was firmly adherent to the structures of the infratemporal fossa. The protruding mass was resected, revealing on the cut surface a pale hyaline-like cartilage. Frozen sections were not made. At this time no further exploration was done.

The pathologist reported a mass, $3 \times 2\frac{1}{2} \times 2$ cm. in size, partly covered by a capsule. It was firm and appeared to be composed largely of cartilage. Microscopic study proved the lesion to be a low-grade chondrosarcoma (Fig. 4).

The same wound was reentered three days later. The incision was extended inferiorly to allow exposure for ligation of the external carotid artery. The remainder of the parotid gland was removed except for its deep pterygoid portion behind the ramus of the mandible. The tumor was found to be encapsulated at its lateral, anterior, and inferior borders (Fig. 8). It was closely adherent to the anterior surface of the temporomandibular joint, the squama of the temporal bone, and the great wing of the sphenoid bone, where no capsule could be found. The tumor also extended to the posterior wall of the maxilla. Medially, it filled the pterygomaxillary fissure and covered the lateral pterygoid plate, causing complete atrophy of the external pterygoid muscle. Inferiorly, the internal pterygoid muscle was found to be partly atrophic; it limited the extension of the tumor in this direction. The neoplasm was found in close contact with the foramen ovale. The pterygopalatine fossa was partly emptied of tumor material. The mass was removed with a curette to the limits of the infratemporal fossa. Brisk venous bleeding occurred, requiring a transfusion of 500 cc. of whole blood. Oxidized cellulose (Oxycel) gauze sponge was placed in the fossa for hemostasis. A Penrose drain was placed in the lower portion of the wound.

Fig. 8 (Case 1).—Model of the chondrosarcoma in the infratemporal fossa.

The postoperative course was uneventful except for some trismus and edema of the eyelids. There was also complete anesthesia along the area of distribution of the mandibular nerve. The facial nerve was intact.

In January, 1955, ophthalmological consultation revealed a borderline increase of the intraocular tension, which later returned to normal. The patient had no pain for two months after the operation. In March, 1955, however, the pain gradually returned and fullness below the left zygoma became apparent. The patient was referred for megavoltage therapy.[34] A 2,000,000 electron volt machine was used at a distance of 125 cm. A half-

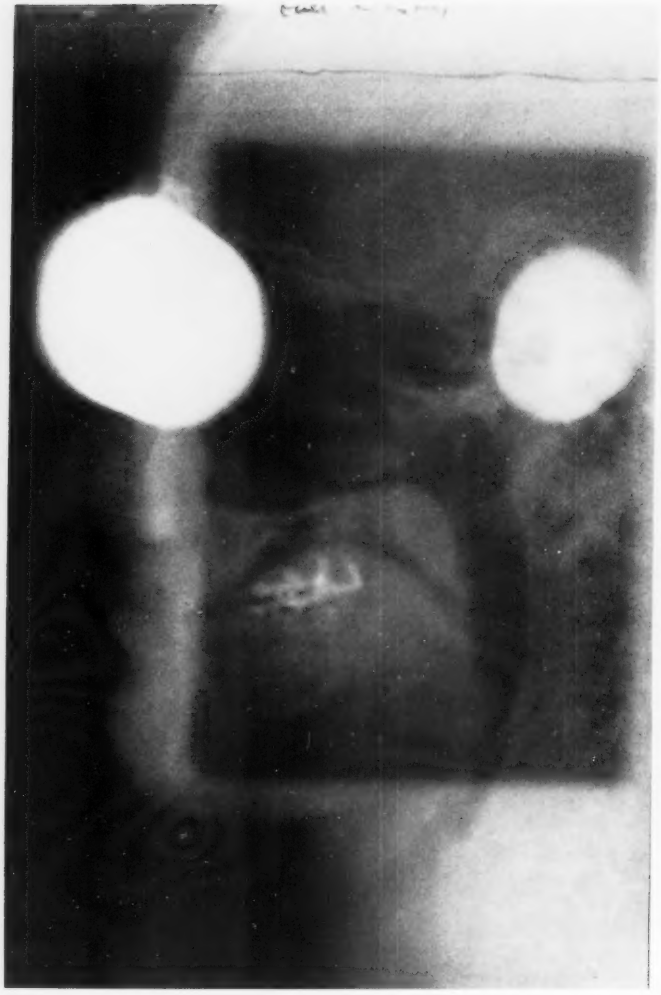


Fig. 9 (Case 1).—X-ray photograph showing the lateral port used for megavoltage radiation therapy. The eye and cartilage of the ear were masked with lead.

value layer of 12.5 mm. of copper was used. An average tumor dose of 180 r a day was given in a series of 33 treatments. From March 25, 1955, to May 18, 1955, he received a total dose in air of 6800 r. The tumor received 6019 r. A lateral port was used on the side of the lesion. The eye and cartilage of the ear were masked from the radiation (Fig. 9).

The need for analgesia gradually subsided. Since then he has been maintained on acetylsalicylic acid several times a day. In February, 1957, a slight fullness of the left nasopharynx and soft palate appeared. Two months later a serous otitis media of the left ear occurred. The Eustachian tube could not be inflated by Politzerization or by catheter. Examination revealed a 15 to 20 db. hearing loss in the affected ear. After myringotomy, there was immediate relief, which did not last. A polyethylene tube was placed through the tympanic membrane. This has been replaced about once a month and has afforded subjective relief (Figs. 10 and 11).

Case 2.—A 31-year-old white woman entered the hospital in March, 1952, for duodenal ulcer. At that time, the right side of her face developed a steady ache at the nasoorbital angle with radiation to the ear. The ache was always in the upper jaw. The pain was thought to be from an infected right lower tooth. This was extracted, giving no relief. A swelling developed over the right side of her face below the zygoma. All of her teeth were extracted. The pain persisted. In December,

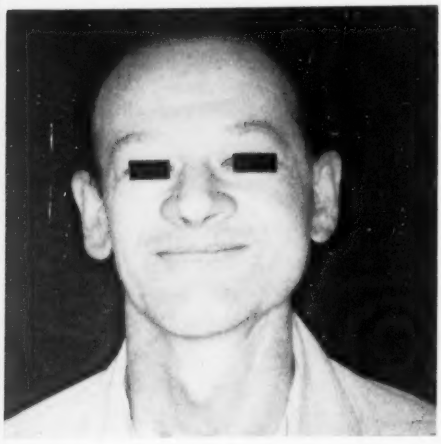


Figure 10

Fig. 10 (Case 1).—Patient with chondrosarcoma of left infratemporal fossa two and one-half years after deep radiation therapy. The asymmetry of the face has remained the same since treatment.

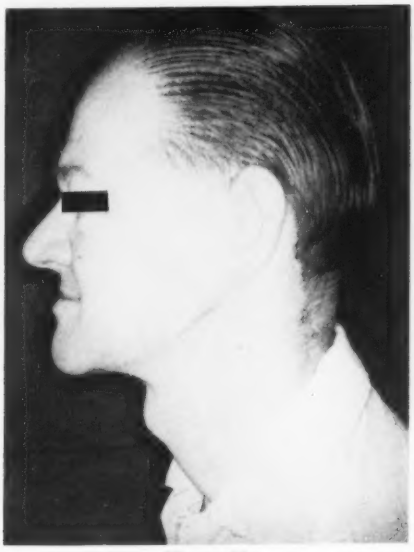


Figure 11

Fig. 11 (Case 1).—Lateral view two and one-half years after deep radiation therapy. The condition of the skin was always good.

1952, she again saw her local physician for the pain and swelling of the right side of her face. A mass was found high in the right maxillobuccal sulcus of the vestibule of the mouth. In January, 1953, iodized oil (Lipiodol) was injected in the duct of the right parotid gland. X-rays of the parotid gland were reported as normal. Iodized oil was injected into the right maxillary sinus in May, 1953. The x-rays were normal. Complete blood count and urinalysis were reported as normal.

In June, 1953, an ovoid mass was removed from the infratemporal fossa by Dr. Myerson.[85] It was approached by means of an incision in the right maxillobuccal recess (Fig. 12). The pathology report was hemangiopericytoma, neurofibroma, or neurofibrosarcoma.

An abscess occurred in the surgical area postoperatively; it was drained 10 days later. During the following three months, partial relief of pain was obtained by cocainization of the spheno-

Fig. 12 (Case 2).—Surgical approach for the patient with neurofibrosarcoma.

Fig. 13 (Case 2).—X-ray demonstrates the extent of the neurofibrosarcoma. The arrows show the extension of the tumor through the posterior wall of the right maxillary sinus.

palatine nerve distribution within the nose. There were intermittent attacks of anesthesia of the right cheek at the corner of the mouth. When the normal sensation returned, the pain was severer. By October, 1953, 19 months after the onset of the tumor, the fullness of the area over the zygoma was again present. X-ray studies revealed a diffuse area of increased density associated with loss of bony detail over the base of the skull in the right pterygoid region. The right sphenoid sinus was diffusely clouded (Fig. 13). There was a shadow of the tumor in the posterior lateral wall of the right maxillary sinus.

The next month a Caldwell-Luc operation was performed. The interior of the right maxillary sinus revealed a large, rounded, reddish, semisolid

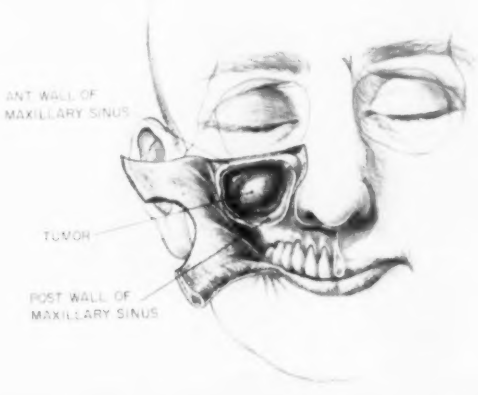


Fig. 14 (Case 2).—Fergusson approach for removal of neurofibrosarcoma from the infratemporal and pterygopalatine fossae. The illustration shows the tumor in place. Further exenteration of the maxilla medially and laterally was done when the tumor was removed.

circumscribed mass. The posterior sinus wall was absent. The mass was adherent to the ethmoid and sphenoid bony walls; it also extended into the infratemporal fossa and was removed from this region. Attempted removal of the tumor from the pterygopalatine fossa caused excessive hemorrhage. A gauze pack was placed in the operative area. The right external carotid artery was ligated, and a transfusion of 500 cc. of whole blood was given. The pathological report was neurofibrosarcoma (Fig. 5).

The patient had considerable relief from pain until January, 1954, at which time pain again became severe in the right side of the face, ear, and occiput.

Another operation was performed in January, 1954. A Fergusson type of incision was made (Fig. 14). A large mass extending to the recesses of the infratemporal and pterygopalatine fossae was removed in several large pieces. A gauze pack was placed in the area, and 1000 cc. of whole blood was given. The pack was gradually removed over a period of 17 days.

The gross specimen consisted of three parts. The largest measured 5×4×3 cm. The microscopic diagnosis was neurofibrosarcoma.

The patient was comfortable during the following eight months. In September, 1954, she developed diplopia and increased trismus. There was bulging of the hard and soft palate in the region of the tuberosity extending to the midline. A whitish mass resembling a polyp could be seen in the right side of the nasopharynx. She had developed Eustachian tube obstruction. There was also displacement of the right eye outward.

Consultation was obtained at the Mayo Clinic in October, 1954. Their pathology department concurred in the diagnosis of neurofibrosarcoma, Grade I. Radiation treatment was recommended.

In October, 1954, palliative x-ray therapy was started.[26] Lateral and anterior ports were used with a total dose in air of 6000 r. These factors were used: 250 kv., 15 ma., filtration 1 mm. copper, and 1 mm. of aluminum, giving a half-value layer of 2.05 mm. of copper at a distance of 50 cm. No significant improvement resulted from the x-ray therapy.

There was progressive diminution of her vision in the right eye. Neurological examination on Jan. 31, 1955, showed moderate pallor of the right optic nerve. The extraocular muscles were intact, but conjugate diplopia was present because of globe displacement. No sensory or motor abnormalities were noted.

The pain persisted so that in May, 1955, a tracheotomy was done in preparation for a craniotomy. The ninth nerve was sectioned. The fifth nerve could not be cut because of an intracranial mass adherent to the pons blocking the approach. She was hospitalized several times later for control of pain.

Fig. 15 (Case 2).—The base of the brain demonstrating several large masses.

On Nov. 26, 1955, a prefrontal lobotomy was done. She died the following day.

Postmortem examination revealed the tumor mass to have invaded the right maxillary sinus, the nasopharynx, the ethmoid and sphenoid sinuses. It had extended as far posteriorly as the foramen magnum. Intracranially, a huge nodular mass occupied the right middle fossa of the skull extending over the midline and into the right cerebellopontine angle. The base of the brain presented a large tumor formed in several large masses (Fig. 15). The main mass was firm, fibrous, and grayish-pink in color. It measured 5.3×4.5×2.5 cm. and involved the right temporal lobe. The second mass measured 2.6×2.8×2.2 cm. and occurred in the interpeduncular fossa. The third mass constituted the cerebellopontine angle tumor and measured 3.7×3.3×3.1 cm. The bifrontal lobotomy incisions were noted. The microscopic examination revealed the brain tumor to be the same as the primary lesion.[27]

Comment

The case reports are similar because of the presentation of the tumors in the infratemporal fossa and the anesthesia of the buccinator nerve. There is further similarity in the later development of Eusta-

chian tube obstruction and growth of the tumor in the nasopharynx and palate.

In the second case of neurofibrosarcoma, the tumor may have originated first in the pterygopalatine fossa. This theory is based on the history of pain over the distribution of the maxillary nerve. After prolonged development, it would later appear in the infratemporal fossa. Proptosis of the right eye occurred after the mass presented in the infratemporal fossa.

Pathology

Grossly, this low-grade chondrosarcoma resembled a clear homogenous hyaline-type cartilage. The microscopic examination was verified by several pathologists, including the Armed Forces Institute of Pathology.

Chondrosarcomas, which are clinically softer and very cellular, should be considered more malignant.[38] Lichtenstein[28,29] gives these microscopic criteria for malignancy of chondrosarcoma: many cells with plump nuclei, more than occasional cells with two such nuclei, any giant cartilage cells with large single or multiple nuclei or with clumps of chromatin.

Neurofibrosarcoma is usually rounded or oval, although it may assume unusual shapes because of varying tissue resistance in the jaws.[31] It infiltrates insidiously, metastasizing freely through the blood stream, but rarely through the lymphatics.[30] Neurofibrosarcomas do not always retain clearly defined histological characteristics, especially when metastasis has occurred. According to Ewing[38] three main groups of neurogenic sarcoma may be recognized. They are the sclerosing fibrosarcoma, the spindle-cell neurosarcoma, and the cellular anaplastic neurosarcoma. Neurofibrosarcoma and neurosarcoma are terms applied to spindle-cell growths which vary in number and size of cells and the amount of anaplasia present, so that we have both the hard fibrous types and the softer, more cellular forms which may show myxomatous changes. A palisade arrangement of the cells is often visible. Interlacing whorls of cells are characteristic of the active forms of this tumor.[38]

This case of low-grade neurofibrosarcoma presented a problem of classification. The first tissue report was hemangiopericytoma, neurofibroma, or neurofibrosarcoma. Later consultation with the Tumor Tissue Registry[39] and with the Department of Pathology at the Mayo Clinic agreed that this was most likely a low-grade neurofibrosarcoma.

Treatment

The surgical cure of malignant tumors of the infratemporal and pterygopalatine fossae may always be considered difficult. Ehrlich[21] successfully removed an encapsulated mixed tumor from this area through an intraoral approach. He mentioned the possibility of an external approach by cutting through the angle of the mandible. Mündnich[40] recently discussed the Fergusson type approach in which the infraorbital incision is extended far laterally, giving exposure to the zygoma and infratemporal fossa. Priest and Kucera[41] recently discussed the virtues of the Fergusson approach for radical surgery of the maxilla. Fischer[42] reviewed the surgical approaches for the pterygopalatine fossa. He listed the transorbital, transzygomatic, transpalatal, and transantral approaches. He felt that the transantral approach is the most satisfactory method of reaching this area.

In the selection of cases for radical surgery, Moffet[43] considered these criteria: The patient should be expected to survive the operation, and there must be a reasonable chance of excising the growth.

The surgical approach in the case of chondrosarcoma was similar to the one used by Pasternack[7] and Gingrass.[6] The procedure was modified by removing the parotid gland in order to preserve the facial nerve by direct visualization.

According to Lichtenstein[28,29] radiation may serve at most as a palliative agent for a chondrosarcoma. It is generally considered that chondrosarcoma is insensitive to

radiation.[23] Dahlin [44] reported 58 patients who received x-ray therapy. Fifteen had some regression, although ultimately the course of the disease was not affected. Harmer [45] reported a case treated with radium needles. The patient was well 10 years later. He gives much credit to the radiation therapy.

Ward and Hendrich,[46] Butterworth,[47] and de Cholnoky [48] feel that radiation therapy is of little or no value in treatment of neurofibrosarcoma.

Prognosis

Chondrosarcoma may start slowly over a 12- to 24-year period.[28,29] Metastasis occur in less than 10% of cases. If the tumor is inadequately removed, the lesion tends to become more invasive with each recurrence.[28,29] Metastasis to the lungs has been reported.[11,28,29] There are frequent references in the literature in which multiple excisions have been done.[11,12,45] In surgically accessible areas, 41% were cured when adequate excision had been done. A 10-year follow-up has been suggested.[44]

Sarcomatous changes take place in 6.6% to 16% of patients with neurofibromatosis. The lesion is at first well encapsulated. With recurrence, it becomes less encapsulated and more infiltrative. Metastases occur late in 25% to 50% of the cases, according to Butterworth.[47]

Summary

The symptoms and physical signs of primary tumors in the infratemporal and pterygopalatine fossae are presented.

The pertinent anatomy, differential diagnosis, incidence, and embryology of these tumors are reviewed.

Two case reports are presented. Their early physical findings and progression of the disease to involve other anatomical areas show similarity. The pathology, treatment, and prognosis are discussed.

Conclusions

Undiagnosed, persistent facial pain of mandibular nerve origin with anesthesia of the distribution of the buccinator nerve is suggestive of a lesion in the area of the infratemporal fossa. Trismus may not be a leading symptom.

Although both of these cases of primary tumors of the infratemporal and pterygopalatine fossae were of low-grade malignancy, the extent of the growth by the time of discovery precluded the hope of cure by surgical attack. X-ray therapy has been only partially successful.

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Audio-Prosthetic Management of Eustachian Tube Blockage

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Often in the practice of medicine the exact order in which a patient relates his symptoms promptly suggests to his physician a specific line of clinical investigation. Approximately three years ago a patient was examined whose complaints were only those of pain in the jaw joint and hearing loss on the same side.

As observed by a younger otolaryngologist, interest in symptoms related to dysfunction of the temporomandibular joint has waxed and waned. Initial enthusiasm was tempered by older confreres and teachers who were pressed to take a stand pro or con. Attempts to explain pinched nerves as producing pain when an intact meniscus was demonstrable by x-ray examination led to further doubts. The more recent concept of disturbed neuromuscular physiology with dental malocclusion caused by missing teeth and prolonged dental microtrauma as etiological factors producing muscle spasm and pain is now widely accepted. An excellent historical review of this entire problem has recently been presented by Freese.[1] Though I was aware that serious doubts regarding any relationship with hearing loss have long existed, further study of this debatable point was prompted by the above patient.

Since 1920, when Wright[2] first related hearing loss to mandibular joint abnormality, many explanations have been proposed for and against such an occurrence. Costen[3] included hearing loss as part of a broad syndrome and related it directly to malfunction of the temporomandibular joint. He pointed out that almost two-thirds of the lateral wall of the Eustachian tube consisted of the tensor muscle of the soft palate, and, when studied in wet specimens, this muscle wrinkled up and obstructed the tube when the lower molar teeth were missing.

Later the same author[4] felt that he could no longer support his previous impression that hearing loss in these cases was due to Eustachian tube obstruction. In his most recent series of 400 cases he pointed out that all of his patients showed a high-tone hearing loss and only those with obvious fluid in the middle ear, and these were very few, evidenced any low-tone hearing loss representative of tubal blockage.

This is a preliminary report of studies on 160 patients with dental malocclusion and is limited to 100 of the same group whose complaint was primarily that of hearing loss. Further to explain the title, the audio portion refers specifically to (*a*) audiometric findings and (*b*) to findings on Eustachian tube auscultation. Prosthetic portion is used in the broadest terms and relates to the armamentarium for the correction of the malocclusion.

Recent studies on an entirely separate group of over 2500 patients indicate that maximal audiometric losses occurring at the 6000 cycle level appear to be associated with Eustachian tube pathology. For example, in dealing with cases of serous otitis media the first tones to be disturbed are at this level and these same 6000 cycle tones are the last to show optimum improvement following clearing of the fluid from the middle ear and mastoid. Further, it has

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been evident that a considerable amount of fluid can be present in the middle ear without producing any significant low-tone hearing loss.

Since the 6000 cycle tone is usually not included as a routine frequency in office audiometry, it is most interesting to note that in all of the present dental malocclusion cases included in the present preliminary report the major loss was invariably at this pitch. The 4000, 8000, and 11,000 cycle tones routinely tested often showed evidence of loss in the present series, but the 6000 cycle tone showed the maximum loss. These findings, however, are not limited to patients with malocclusion but appear to be evident in all situations where there has been infringement on the patency of the Eustachian tube. Mucosal swelling primarily on an allergic basis, with or without superimposed upper respiratory infection, is a far commoner cause. It therefore appears that not only is a high-tone conduction hearing loss a definite clinical entity but that this may explain why Costen was unable to finally prove a relationship to Eustachian tube blockage.

Eustachian tube auscultation, like the field of otolaryngology, is both an art and a science. In this sequence ability lags far behind the young house officer's ability in performing a tonsillectomy. In examining these patients it gives much evidence of Eustachian tube and middle ear pathology that Politzerization masks or condenses into a fraction of a second. Although the fit of the catheter into the pharyngeal orifice cannot be as precise as one would wish because of septal deviations causing slight changes of the angle of the catheter tip against the walls of the orifice, yet significant changes are easily detectable by the gentle-handed and careful listener. Even the position of the patient's lower jaw is important in interpreting the sound of air passing through the Eustachian catheter and the Eustachian tube. The length of delay in hearing the sound of the tube opening, the pitch of the sound of air passing through the tube, the presence of moisture in the tube, the presence of fluid in the middle ear with a harsh, rough auscultatory sound, or lub-blup of air passing through fluid, or even no sound at all with a full middle ear and mastoid are all of diagnostic help [examples on tape recording].

Eustachian tube auscultation in all 100 patients showed evidence of narrowing on the side of the hearing loss associated with dental malocclusion. In many instances, this was bilateral, and the narrower side was always associated with the greater hearing loss.

Patient Examination

Several additions to routine office ear, nose, and throat examination have been found helpful in suspecting malocclusion as a cause for deafness.

1. During nasopharyngoscopy with the nasopharyngoscope both degree of patency and excursion of the torus on wide opening of the patient's mouth and on "biting down hard" is noted. Narrowing and limitation are suggestive of malocclusion.

2. Palpation of the temporomandibular joints anterior to the tragus is performed. The patient is instructed to open the mouth slowly and then to close slowly. Asynchrony of condylar motion is noted, evidence of crepitus is sought, and the phase of opening during which it occurs is recorded. Deviation of the mandible from the vertical is noted and often presents an S or reverse S-shaped conformation.

3. Auscultation with the stethoscope bell over the joints will confirm and pick up the easily recognized pathological click of the meniscus suddenly being drawn forward by the superior head of the external pterygoid to which it is attached.

4. Bilateral joint palpation with the examiner's middle fingers in the patient's external auditory canals will reveal evidence of incoordinate forward condylar motion.

5. Evidence of premature contact of two opposing teeth, an early finding, is sought. The patient is asked to swallow. During this the upper and lower teeth come together and then separate a short distance, bringing the mandible into the "position of rest." As the patient slowly approximates the jaws from this position, the point of initial contact becomes evident. More muscle tension is necessary to bring the remaining teeth into con-

tact. Repetition of this several times will verify the premature contact. Finger palpation along the buccal aspect is also confirmatory.

6. Initial audiometry precedes Eustachian catheterization so that the latter does not mask any of the high-tone loss or low-tone loss.

7. Speech audiometry is frequently helpful in further establishing a primary conduction loss, although air and bone figures may strongly suggest a mixed and primarily perceptive loss.

8. Recheck air and bone studies following catheterization may show striking improvement in the loss initially noted.

As would be expected from Costen's earlier work, this condition is much more frequent in the older age groups, where missing teeth and the presence of partial and full dentures are so commonly found. In younger persons the changes are milder; the degree of hearing loss is less, and primarily above the speech range; yet for the sake of conservation of hearing attempted correction of the pathology is most worth while.

Sources of emotional tension with resultant clenching of the jaws increase the muscle spasm and are reflected directly in the wear patterns of occlusal surfaces of the teeth. Specific attention to identification of these emotional tensions is necessary in the evaluation of each case. The otolaryngologist and the dentist in this correction must be prepared to deal effectively. In some instances psychiatric consultation will be found advisable.

In addition to muscle relaxants, such as meprobamate (Equanil) and mephenesin carbamate (Tolseram), and selective contouring of teeth, the further armamentarium used in other cases in this series included, when indicated, temporary splints, muscle exercises, orthodontia, replacement of missing teeth either with permanent bridges, replaceable partial or full dentures, and in every instance a detailed discussion with the patient of the present understanding of the mechanics causing his particular difficulty.

Many otolaryngologists have referred patients back to their own dentists for the relief of pain from temporomandibular joint dysfunction, and the dentist's reply is that he hopes he never sees another joint problem. In our locale the well-trained periodontist who has extended his interests to embrace the fields of oral medicine and the patient as a whole is now a major member of the team handling otolaryngological problems related to malocclusion. Dentists in your community with this broad background will be most interested in helping with problems of this type. Further training is readily available if desired in some of the largest centers.

Summary and Conclusion

A preliminary report on consistent findings in 100 patients with dental malocclusion and hearing loss is presented. Two previously unreported findings were present in all of the cases. (1) Maximal 6000 cycle hearing loss. (2) Auscultatory evidence of Eustachian tube narrowing, the degree of narrowing corresponding with the side of the greater loss. These findings indicate that Costen's inclusion of hearing loss as a symptom in his temporomandibular joint syndrome was initially correct, although he later withdrew it since he expected the blockage to be represented by low-tone loss. A list of otolaryngological office examination clues to dental malocclusion is given. Management by a dentist with a broad background embracing the field of oral medicine is indicated.

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Anatomy of the Bronchial Tree

A Presentation of an Extension of the Jackson-Huber Nomenclature of the Tracheobronchial Tree

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For the past two and a half years we have been using general anesthesia for peroral endoscopy at the Long Island College Hospital. This method has as its basis the use of an ultra-short-acting intravenous barbiturate, an extremely short-acting muscle relaxant (succinylcholine), and a mechanical thoracoabdominal respirator. A description of this method of controlled passive respiration under general anesthesia is the subject of a separate paper. As a result of this type of anesthesia we have been able, in a series of approximately 200 cases, to visualize more distal bronchi than is usually possible with topical anesthesia. This is so because the patient is asleep, completely atonic, and is being adequately ventilated by the respirator. Therefore, the need for haste is eliminated; motion at the head and neck is possible through a wide range; the normal physiologic motion of the bronchi is present, and cough is absent; consequently, it becomes possible to carefully insert the bronchoscope more distally than is usually the case.

We should like to make is clear that it is not possible to see all of the additional bronchi described later in this paper in every case. It has been our experience, using this method of general anesthesia, that in approximately 40% of cases, at least some of these more distal bronchi can be seen. In some cases many of them can be visualized, and in others, very few. When referring to Figures 7 and 8, v. i., the reader is therefore asked to bear in mind that this represents a composite of many cases. Not all of the bronchi indicated as being an extension of the present nomenclature can be seen in any one case. However, in a series of cases all of these additional bronchi can and have been seen.

Two more factors must be mentioned: (1) The size of the bronchoscope; (2) The anatomy of the bronchial cartilages.

With regard to the size of the bronchoscope, in a routine adult bronchoscopy we had been using a 8 mm.×40 cm. distally illuminated Jackson bronchoscope. However, we found that the 8 mm. bronchoscope limited entry into distal bronchi. A 6 mm. bronchoscope, 40 cm. long, permits entry into the right upper lobe bronchus and in some cases into its anterior and posterior segmental orifices, into the left upper lobe bronchus and frequently into its lingular orifice, into the middle lobe bronchus, and into the segmental orifices of both lower lobe bronchi. Furthermore, the 6 mm. bronchoscope will admit the Broyles Optical bronchoscopes, Foroblique, right-angle, and retrograde. Inasmuch as the small size bronchoscope allows adequate air exchange around the bronchoscopic tube, one need not be concerned about obstruction of the bronchoscope by the telescope. Moreover, our technique permits complete relaxation of the larynx and a wide glottic chink to further the security of adequate ventilation.

Before discussing the second factor, that is, the anatomy of the bronchial cartilages,

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it might be well to review very briefly certain terminology:

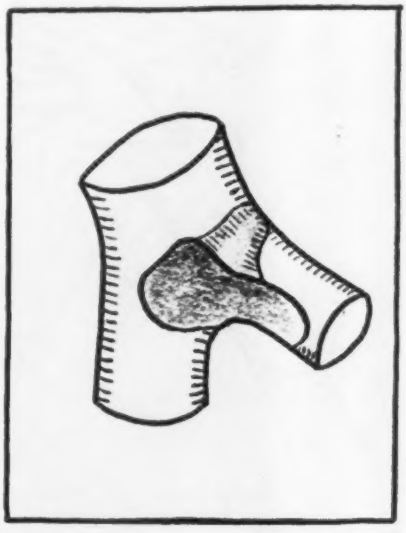
The term primary bronchus refers to the right and left main bronchi; the term secondary bronchus refers to the following: right and left upper lobe bronchi, right middle lobe bronchus, and right and left lower lobe bronchi. Tertiary bronchi are the segmental divisions of the secondary bronchi, a few examples of which are the following: anterior, apical, and posterior bronchi of the right upper lobe, or upper and lower (or lingular) divisions of the left upper lobe bronchus. These tertiary bronchi, or segmental bronchi, constitute the end-point of the present Jackson-Huber nomenclature. Certainly this classification has been of tremendous value in localizing lesions anatomically so that all concerned—bronchoscopists, thoracic surgeons, and radiologists—have been oriented. Beyond these are quaternary bronchi.

To return to the importance of the anatomy of the bronchial cartilages, anatomical dissections have shown that the orifice of a bronchus may be formed of a saddle-shaped or semilunar cartilage (i. e., an incomplete ring), which is the case in most of the secondary bronchi; or it may be a complete ring, which usually characterizes the orifices of the tertiary and quaternary bronchi. When the orifice is formed by a saddle or semilunar cartilage, flexibility and elasticity are naturally increased (Figs. 1, 1A, and 2).

If a 6 mm.×40 cm. bronchoscope is introduced, the trifurcation of the right upper lobe bronchus, or the bifurcation of the left upper lobe bronchus can be actually reached fairly frequently. Then the use of an optical bronchoscope may permit additional examination of the subsegmental or quaternary bronchi. In a number of cases the bronchoscope can be inserted into the entrance of the anterior and posterior segments of the right upper lobe bronchus, or into the lingular division of the left upper lobe bronchus. This, in combination with a suitable optical bronchoscope, makes it possible to view the orifices of quaternary bronchi. Similarly, the introduction of a small bronchoscope into the middle lobe bronchus demonstrates the tertiary and often the quaternary bronchi. The segmental orifices of the lower lobes can be entered in some

Fig. 1.—Schematic drawing showing a bronchial orifice being formed by a ring-shaped cartilage.

Fig. 1A.—Schematic drawing showing a bronchial orifice formed by a saddle-shaped cartilage (incomplete ring).



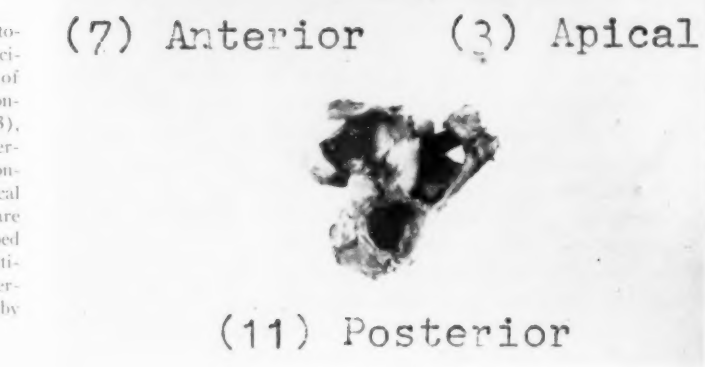


Fig. 2.—Actual photograph of a dissected specimen of the trifurcation of the right upper lobe bronchus into its apical (3), anterior (7) and posterior (11) segmental bronchi. Note that the apical and anterior bronchi are formed by saddle-shaped (incomplete ring) cartilages and that the posterior bronchus is formed by a complete ring.

cases and this allows not only more thorough examination of these, either directly or through a suitable optical bronchoscope, but in addition we are able to visualize orifices and parts of intrasegmental or subsegmental, quaternary bronchi beyond (Figs. 3 to 6).

It stands to reason that if more distal portions of the bronchial tree can be visualized, (1) more pathology will be seen earlier, and (2) more definite localization of this pathology can be accomplished.

The demonstration and visualization of subsegmental or intrasegmental bronchi call for accurate description and nomenclature. This paper is concerned with proposing an extension of the Jackson-Huber nomenclature to include quaternary bronchi, which might also be termed subsegmental or intrasegmental. Beyond these we are dealing with bronchioles. Thorough bronchoscopic and anatomical studies have been made, together with a review of the pertinent litera-

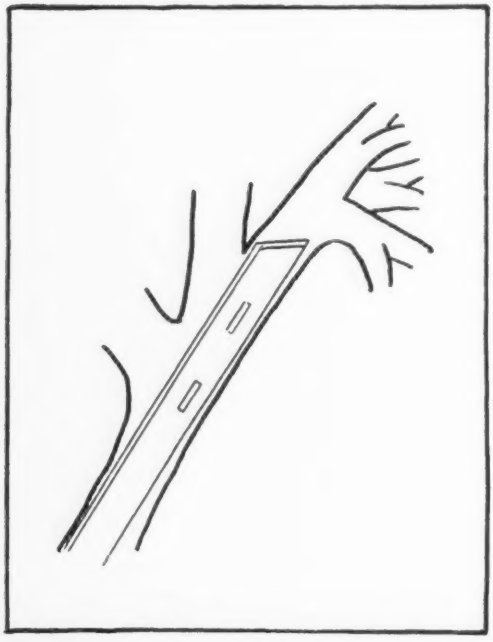


Fig. 3.—Schematic drawing showing bronchoscope within the orifice of the right upper lobe bronchus permitting direct examination of anterior and posterior segmental orifices and some of their subsegments.

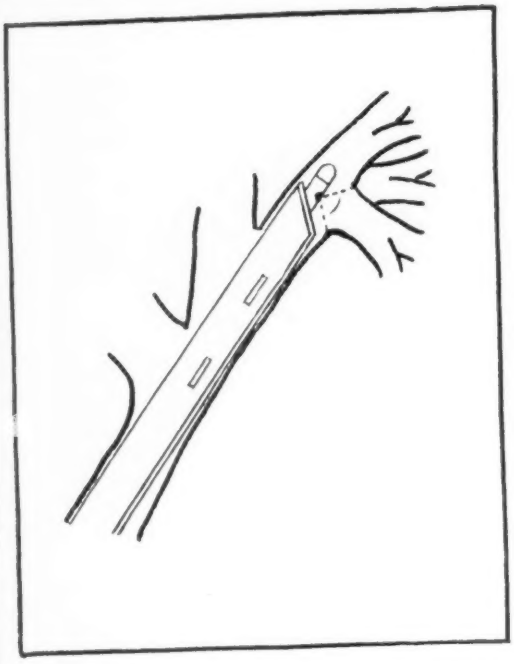


Fig. 4.—Rotation of the bronchoscope and insertion of a right-angle telescope, which brings the apical bronchus and its subsegmental branches into view.

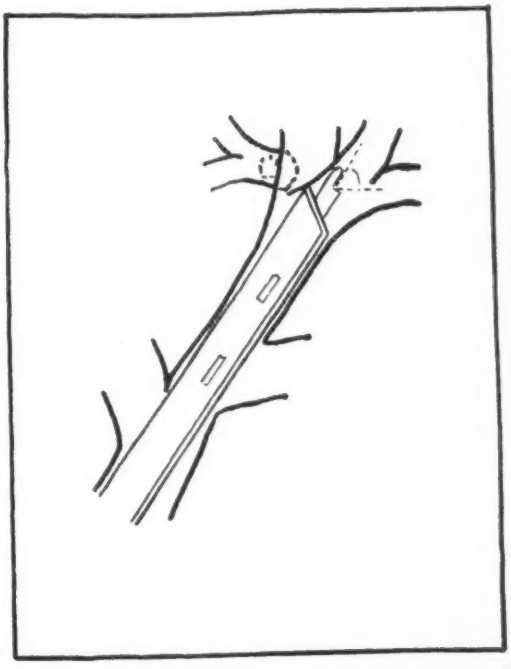


Fig. 5.—The bronchoscope is within the orifice of the right middle lobe bronchus, permitting direct examination of its medial and lateral branches. More detailed examination of these and their subsegmental branches may be possible through a Foroblique telescope.

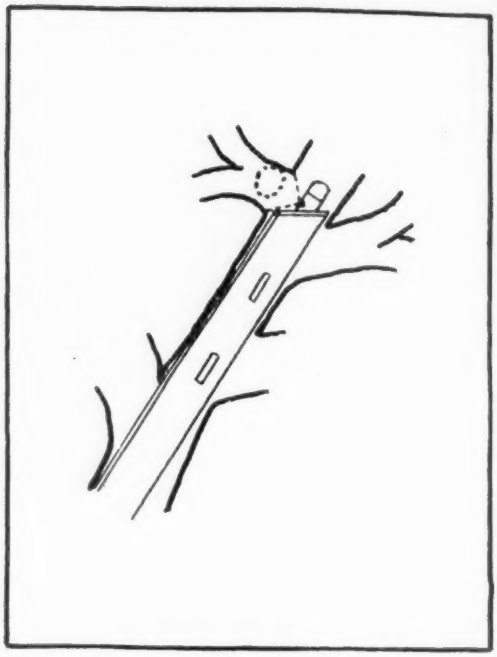


Fig. 6.—The bronchoscope is just proximal to the apical segment of (either the right or left) lower lobe bronchus. A right-angle telescope permits examination of its subsegmental branches.

ture. It should be borne in mind that anatomical variations in position become more frequent as one approaches the periphery of the bronchial tree.

Our proposed extension of nomenclature, therefore, has been based upon endoscopic views using the long axis of the tertiary bronchus as the reference point in naming the quaternary bronchi into which it divides. Hence, anterior and posterior, medial and lateral, superior and inferior, refer to principal directions taken from the long axis of the tertiary bronchi.

Figures 7 and 8 present our proposed extension of the nomenclature of the bronchial tree of Jackson and Huber. The endoscopic appearance is shown in the form of drawings in Figures 9 to 13.

Summary and Conclusions

We feel that any method which permits the discovery of pathology more distal and therefore earlier is of self-evident value. Since it has been possible for us, utilizing general anesthesia with a thoracoabdominal mechanical respirator, to visualize subsegmental or quaternary bronchi, it therefore becomes necessary to name these bronchi.

Mrs. Elizabeth Cuzzort, Director, Department of Medical Illustrations and Photography, prepared the original colored endoscopic drawings.

250 Martense St. (26).
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Figs. 7 and 8.—Schematic drawings of right and left bronchial trees representing the present Jackson-Huber nomenclature (in blue) and the authors' proposed extension of this nomenclature (in red), with correlated lateral and mediastinal surface projections of the subsegments. Note that the numbers in parentheses and their colors (red or black) remain constant in all illustrations (see Figs. 2, 9, 10, 11, 12, and 13).

Figs. 9-13.—Composite drawings of endoscopic views obtained either directly or through various telescopes as indicated in parentheses beneath each drawing. The numbers and colors in parentheses correspond with those in Figs. 7 and 8.

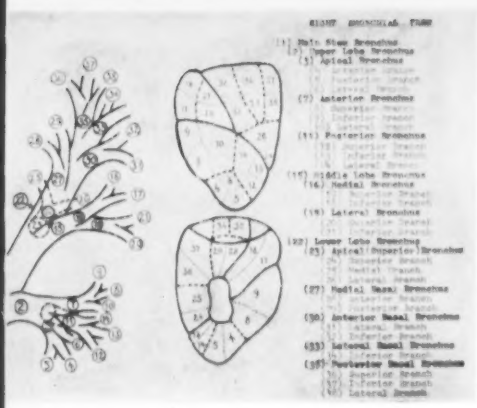

Figure 7

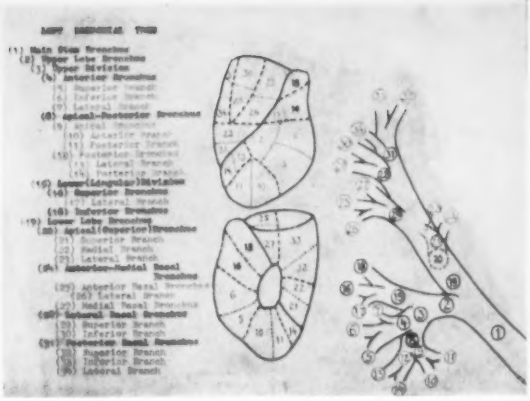

Figure 8

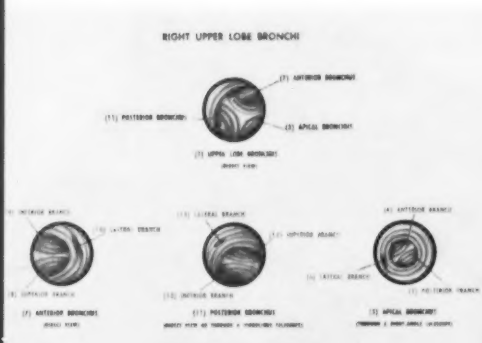

Figure 9

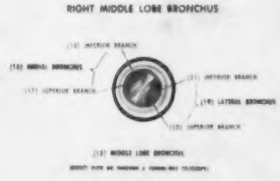

Figure 10

Figure 11

Figure 12

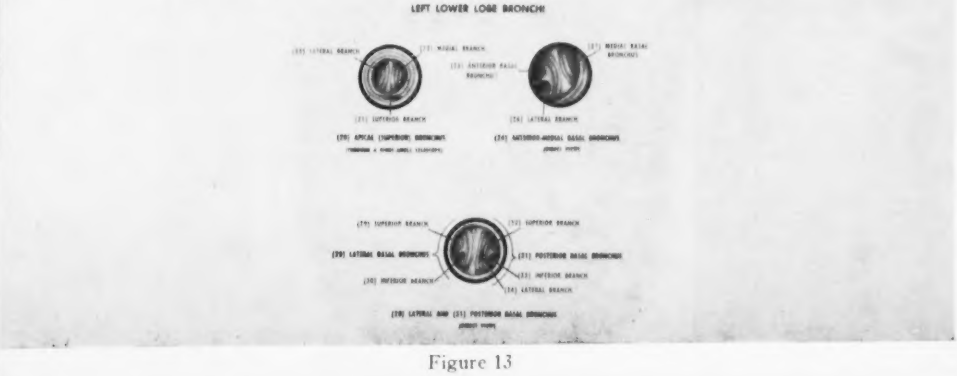

Figure 13

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Postmortem Changes in the Vestibular and Cochlear Receptors (Guinea Pig)

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Autolysis is initiated immediately after death. Since Nager and Yoshii [1] called attention to postmortem changes and the fact that they appear in the inner ear before they are observed in other structures, intravital fixation has been adopted as the best procedure for histological preparations in experimental animals. Nevertheless, confusion about what is meant by normal inner ear still exists. In addition to artifacts, agonal and postmortem changes, position, size, and shape of cells and membranes in living and fixed tissue have been the center of many debates.

The investigation to be described here was primarily an attempt to obtain more knowledge about the sequence of autolytic changes of the inner ear in the guinea pig, as seen under light microscopy. During the histologic study of the guinea pig temporal bones, three additional points become apparent: first, to report the pathologic changes which may be found in young, normal guinea pigs; second, to define the implications and what is meant by normal inner ear; third, to describe the commonest nonpathologic changes which may appear in temporal bones well fixed and stained, the so-called artifacts.

Methods

For the experiments, 81 young healthy guinea pigs were used. The animals were divided into three groups. In Group 1 (13 animals) the temporal bones were fixed by "intravital fixation" with Heidenhain-Susa solution according to the technique described elsewhere.[2] The amount of saline perfused was varied so that it might be possible to find out whether the quantity of saline may alter the inner ear structures. In Group 2 (24 animals) "postmortem perfusion" was done at intervals from 10 minutes to 20 hours after the trachea was clamped. Perfusion was followed by decapitation, dissection of temporal bones, and immersion of the specimen in fixative. In Group 3 (44 animals) the procedure was dissection of temporal bones at different intervals after death and immersion in fixative. This procedure we call "postmortem immersion." However, some variations were adopted. In one set of 11 animals the procedure was decapitation, dissection of temporal bones, and immersion. The time between decapitation and immersion varied from two minutes to three hours. In another set of 23 animals the trachea was clamped, the temporal bones removed from 40 minutes to 20 hours after death and immersed in fixative. In a third set of 10 animals the procedure was as in the previous set, but the right stapes was removed.

In all the guinea pigs both temporal bones were removed and treated in the one block according to the technique described elsewhere.[3,4] In Figure 1, midmodiolar section of guinea pig and cat temporal bones are illustrated. The technique, suitable for small animals, has many advantages over the individual preparation of single temporal bones. It is economical of material; time for sectioning and staining is cut by half, and storage space is conserved. Since both cochleas are cut in approximately the same plane, any point in one is spatially identical with its counterpart in the opposite side, and thus comparison is facilitated and time is saved.

In all the animals Heidenhain-Susa solution was used as fixative. The routine method of hematoxylin-eosin staining for

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Fig. 1.—Results obtained by the technique of removing and staining both temporal bones in one block. (Upper) Guinea pig (×6) midmodiolar section of both cochleas, including the central nervous system. (Lower) Cat (×4).

Notice that the symmetry between the two sides is such that any given region on one side is approximately identical with its counterpart on the opposite side; thus comparison between the two ears is facilitated.

temporal bone was followed. The method gives excellent results when employed by well-trained, experienced technicians; however, precipitates, blood, and cells in advanced autolysis are not clearly differentiated. To overcome this difficulty, chosen sections were stained by Mallory's azan method.

The animals were maintained at room temperature until the temporal bones were removed. Only one animal was kept in a refrigerator (4 C) for 20 hours.

Results

A. Pathological Findings.—Atrophy of the stria vascularis was identified in 16 out of 162 ears. The atrophy was either unilateral or bilateral, localized in the fourth turn (13 cases) or extending down to the second turn (3 cases). The cause of this

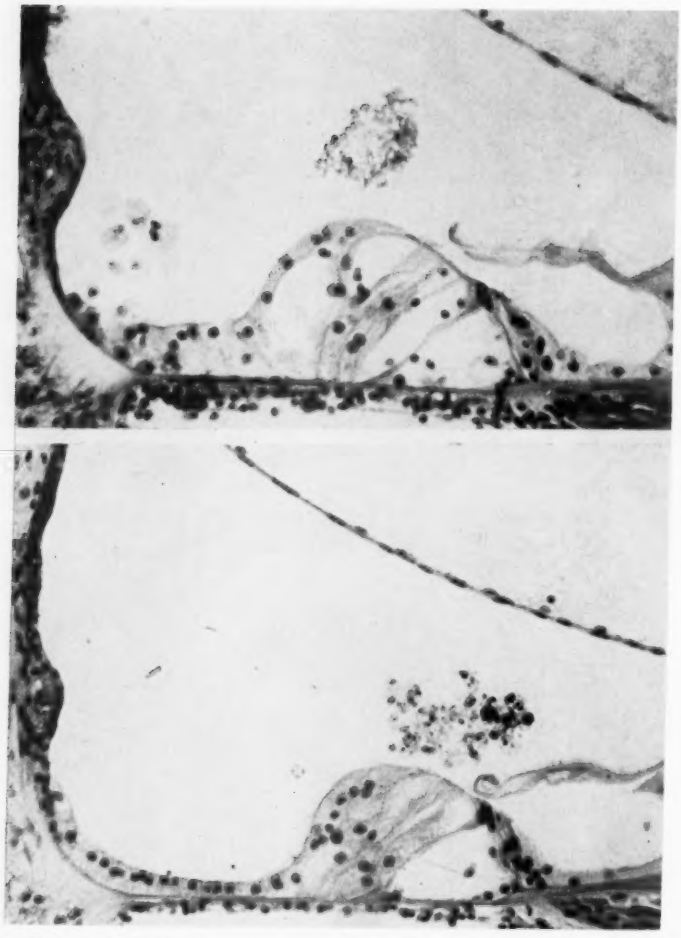

Fig. 2.—Guinea pig B-22. Degenerative process localized in the second and third turn of the cochlea. (Upper) Middle of the lesion, showing atrophy of the stria vascularis, degeneration of the external sulcus cells, some of them detached, and macrophages infiltrating the mesothelial cells. Some cellular infiltration and dilated vessels were found in the spiral ligament. (Lower) Other region, showing atrophy of stria vascularis, organ of Corti in degeneration, and deposits of pigment and cell debris in scala media. Hematoxylin-eosin; × 260.

lesion is uncertain and its effect on the function is questionable. Localized atrophy of the stria vascularis with degeneration of the organ of Corti was found in one guinea pig. Figure 2 illustrates the lesion of 5 to 6 mm. in length, localized in the second and third turn. The nuclei of the external hair cells were pyknotic or absent. Some external sulcus cells were edematous, others detached. Macrophages infiltrated the mesothelial cells and a considerable amount of pigment floated in scala media. These features suggest early stages of a vascular accident of some sort (vasospasm, thrombosis).

The lesion resembled the syndrome of "angiosclerotic inner ear degeneration" described by Saxén.[5] The histopathology of this syndrome showed angiosclerosis of the blood vessels, atrophy of the stria vascularis, degeneration of the organ of Corti and collapse of the cochlear duct. The lesion usu-

Fig. 3.—Guinea pig B-55. Normal microscopic cytoarchitecture of the cochlea as seen through a midmodiolar section (× 19). Intravital fixation. Reissner's membrane was straight but in the basal turn, close to round window, it was undulated. Tectorial membrane remained in contact or near organ of Corti. Hematoxylin-eosin staining.

ally extended along the whole cochlea. In other cases, infarcts or localized lesions were found. These localized lesions presented cystic formation and atrophy of the stria vascularis, either with or without degeneration of the organ of Corti. As Saxén [5] pointed out, infarcts and angiosclerosis may be the cause of localized hearing loss or deafness over the whole range of frequencies.

Except for the reported lesions, no pathology was found in our series of experimental animals.

B. Cytoarchitecture of the Inner Ear.—The distribution, shape, size, and other features of cells, membranes, and fluids of the inner ear have been fully described in the literature. As an illustration, in Figures 3 and 4 the cytoarchitecture of the midmodiolus section of the cochlea, the organ of Corti, the crista with the cupula, and a region of macula utricularis with the otolithic membrane are presented. The result is consistently reproduced, provided the method of fixation and staining is not only well standardized but rigorously followed. Otherwise different results are obtained, as has been well demonstrated by Werner.[6] The whole picture of the inner ear—partially illustrated in Figures 3 and 4—which is obtained after intravital fixation, we labeled the "normal microscopic cytoarchitecture of the inner ear." Important implications are involved in the use of this label, which sometimes are forgotten and consequently give rise to misunderstandings.

First of all, the cytoarchitecture as seen under light microscopy and obtained by a particular method of fixation and staining is not like that of normal living tissue. Fixation, decalcification, dehydration, etc., induce profound changes in the cellular elements, fluids, and membranes. From basic principles of cytological technique [7] it is accepted that the proteins are precipitated in a coarse, sponge-like network. The large

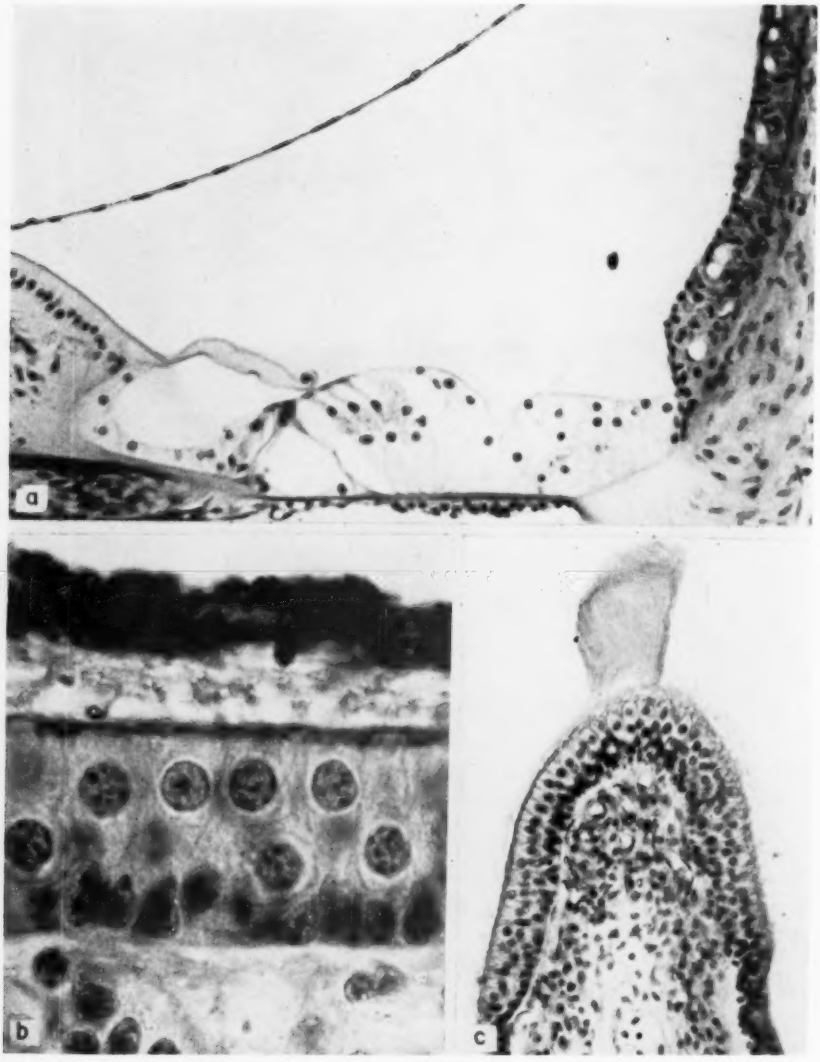

Fig. 4.—Normal microscopic cytoarchitecture of (*a*) organ of Corti (× 240); (*b*) macular utricularis (× 1260); only a small region is shown), and (*c*) crista of one semicircular canal (× 240). Intravital fixation. Hematoxylin-eosin staining.

spaces thus formed become filled with the mounting medium. The centriole may remain, while mitochondria and Golgi's elements disintegrate. Whether disintegration of these structures occurs depends upon the choice of fixative and on subsequent treatment. In the nucleus, the histone is also precipitated as a sponge-like network, and lumps of nucleic acid become trapped among the threads. The karyosomes may remain unchanged, as does the plasmosome. Furthermore, each method of fixation and staining produces specific changes, so that some cytoplasmic and nuclear structures are emphasized. These general principles of cytology apply as well to the inner ear. In our series, intravital fixation with Heidenhain-Susa solution and staining with hematoxylin-

eosin differentiated selectively the nuclear structures of the cell population of the inner ear. All other cell constituents were absent, except the tigroid substance in ganglion cells. Even in the nucleus, differentiation between karyosomes and lumps of nucleic acid remained uncertain. In sections of 16μ-18μ, the cytoplasm appeared as a granular-like network which we suppose is the precipitated proteins. As the sections are made thicker, the network superimposes, and thus the cytoplasm appears more uniform. It must be understood that by cytoplasm we mean the specific artifact introduced by the fixative, that is, the precipitated proteins which make the granular-like network. Therefore, the standard cell in our routine histological preparation shows elements which are not present in the living cell (precipitated proteins) and others which are present (mitochondria, centriole, Golgi's elements) do not appear. The size, shape, position of the nucleus, and other details vary from one class of cells to another. Thus we must define a standard for each one of them, with which any other cell of the same class may be compared. Theoretically, for a particular method of fixation and staining, the standard should be the same from one laboratory to another. Practically, the results vary, not only among technicians, but for a particular one variations may be found from one specimen to another. This occasionally is forgotten and consequently brings about misunderstanding and criticism between observers.

In the living animal, the tectorial membrane covers the organ of Corti, and the cupula extends from the crista to the opposite wall of the ampulla. After fixation and staining, shrinkage and deformation of both tectorial membrane and cupula is constantly found (Figs. 3 and 4). These well-known artifacts we include in our normal microscopic cytoarchitectural picture. Also, the tectorial membrane should remain in contact with or near the organ of Corti. The position of Reissner's membrane should be predominantly straight, although some deviations to either scala vestibuli or scala media are common. Falbe-Hansen [8] found the position of Reissner's membrane was predominantly deviated from a straight line. We think the difference may be due to the procedure of fixation. Falbe-Hansen used Wittmaack's fixative while we employed Heidenhain-Susa's. Whether deviations of a few microns from ideal straight line are significant we cannot decide with the data available. However, in the basal turn, Reissner's membrane consistently is deviated in one way or another and very often is undulated.

Although each method of fixation and staining has its own characteristics, the observer acquainted with them may have some idea of what the normal cytoarchitecture of the inner ear in the living animal should be. We think it is not yet sufficiently emphasized that whatever method of fixation and staining is used, the technique should be mastered, and the observer must know what the procedure does to the living tissue.

C. Artifacts.—In histology, artifact means any change in the cell tissue produced by fixation and staining. In the cytoarchitecture of the inner ear, two types of artifact, primary and secondary, should be differentiated. By primary artifact we mean the results of reaction between reagents and constituents of the cells. This result we defined in Section B as the "normal microscopic cytoarchitecture of the inner ear," and it depends upon the condition of the tissue before fixation and upon the rigorous follow-up of a particular histological method. By secondary artifact, we mean any change which may appear in the normal microscopic cytoarchitecture, defined above. The changes appear because the condition of the tissue was not what we thought to begin with, and/or, intentionally or unintentionally, there was some variation in the procedure. Even a change in brand of some reagents may be critical. In subsequent pages, by artifact we shall always mean, unless otherwise specified, the secondary artifact. We shall describe only those which

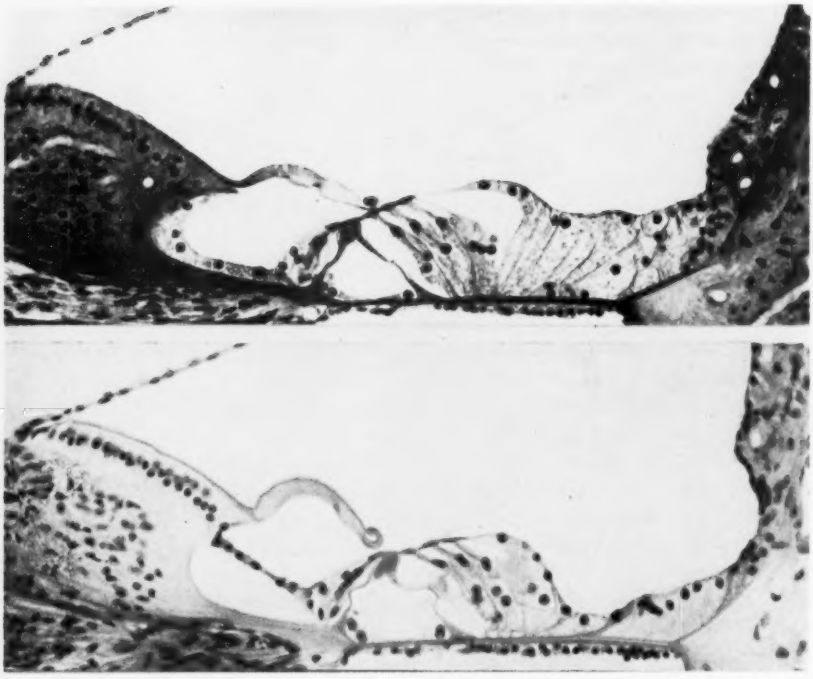

Fig. 5.—Normal microscopic cytoarchitecture of the organ of Corti as given by intravital fixation with Heidenhain-Susa solution (upper) and Wittmaack's solution (lower), and staining with hematoxylin-eosin. Fixation with Wittmaack's solution gave the characteristic detachment of the internal sulcus cells, and in general the cell populations are more slender than those fixed with Heidenhain-Susa solution. In addition, the angle of sectioning contributed to the voluminous appearance of cells.

may appear in temporal bones fixed with Heidenhain-Susa solution and stained with hematoxylin-eosin or Mallory's azan.

Artifacts such as distortion of structures, ridges, wrinkles, precipitates, etc., may be found not only in the procedures mentioned above but in any histological method. These changes are easily detected and the cause recognized.

Some changes are primary artifacts in one histologic method while in another they are secondary. For instance, detachment of the inner sulcus cells is a primary artifact in specimens fixed with Wittmaack's fluid (Fig. 5) and consequently should be included in the normal microscopic cytoarchitecture of that procedure, while in specimens fixed with Heidenhain-Susa solution, this is a secondary artifact. Animals killed by asphyxia or decapitation always presented hemorrhages in the cochlear aqueduct and inner meatus, and in many cases, in the scala tympani near the round window. These hemorrhages are nothing other than blood which ran into these passages during dissection. They never were found in the cochlear duct or in animals killed by intravital fixation or postmortem perfusion. Therefore, it is an artifact induced by the procedure and consequently should be considered a primary artifact, although it is not related to fixation and staining. In some cases, the serial sections showed that bleeding may occur at the borders of attachment of the round window membrane. The cause of this hemorrhage is obscure. Sometimes, blood appears all along the cochlea; sometimes it is localized in the apical turns. Whether this is artifact induced by the procedure, or pathology, is a matter for careful consideration.

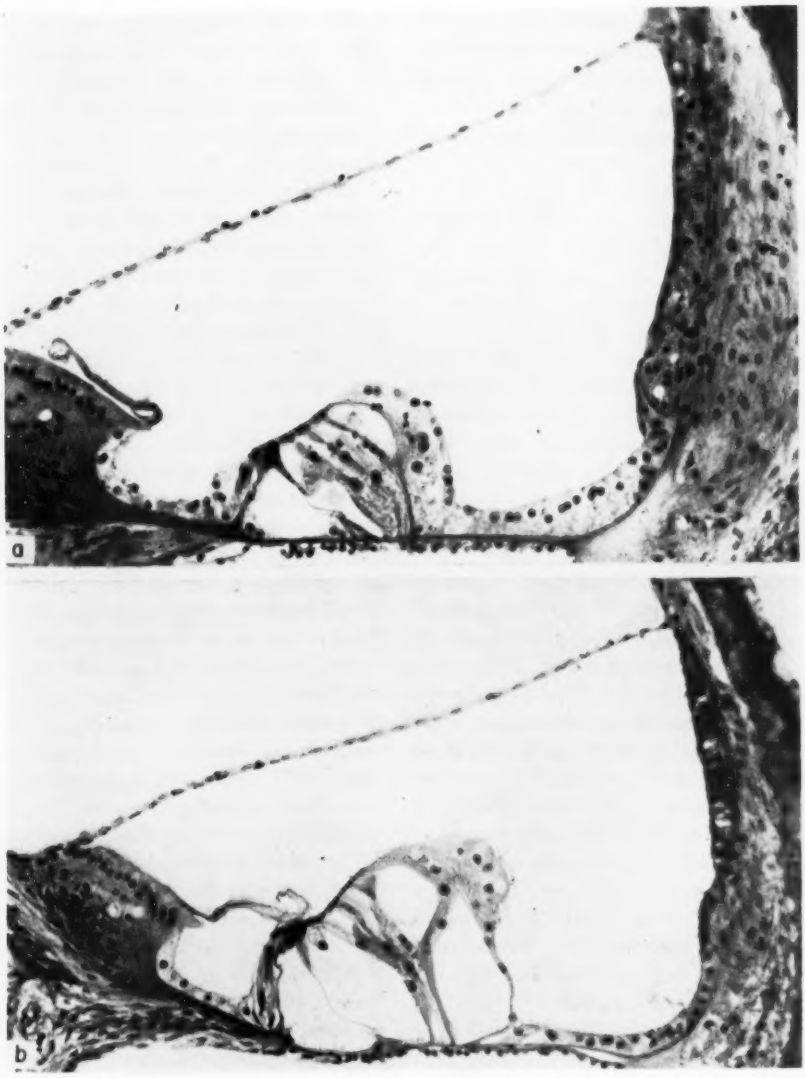

Fig. 6.—Artifacts in the organ of Corti. (*a*) Guinea pig B-17, intravital fixation. Notice perfusion was incomplete in stria vascularis. Tectorial membrane was detached in this region. Precipitates of mercuric chloride in organ of Corti, internal sulcus cells, and spiral ligament. This artifact is due to insufficient treatment with alcohol-iodine. (*b*) Guinea pig B-19, intravital fixation. Detachment of Hensen's from Deiters' cells, and these partially detached from basilar membrane. These space formations were the largest found in our series. The region shown is the apical turn. Both microphotographs stained with hematoxylin-eosin; × 240.

The commonest artifacts were as follows: 1. Detachment of tectorial membrane (Fig. 6 *a*). It was present in almost all specimens fixed by postmortem immersion, either in some spots only or more extensively; less frequently in postmortem perfusion, and only occasionally in those fixed by intravital perfusion. 2. Detachment of Hansen's from Deiters' cells in the apical turns (Fig. 6 *b*). This artifact may be unilateral or bilateral, localized in one spot, or extensive to the third and fourth turns. It was always

related to incomplete intravital fixation or to incomplete postmortem perfusion and was usually present in postmortem immersion. 3. Similarly, incomplete intravital fixation or incomplete postmortem perfusion may give rise to space formation in the internal and/or external sulcus cells. As a rule, the frequency, extension, and size of the spaces increased with postmortem immersion. Mygind, Andersen, and Arnvig[9] reported these changes and lifting of the Deiters' cells from the basilar membrane as "cystic formation." Apparently these authors used the same technique as ours, that is, fixation by immersion in Heidenhain-Susa solution and staining with hematoxylin-eosin. However, this artifact seems larger in their illustrations than ours. This is a good example of variation of results from one laboratory to another even though it seems that the same technique was followed. We prefer to call these artifacts "space formation" rather than "cystic formation" because the latter may be misinterpreted to mean a pathologic condition. 4. The membranous walls of the vestibule generally kept their normal position, but occasionally collapse of the walls over one or more receptors may be found. The set of animals with stapes removed usually presented collapse of the vestibular walls and in some cases, of Reissner's membrane.

Changes in the position of Reissner's membrane have been discussed frequently in the past and the subject has been the center of many theories and debates. Our standard for the three procedures of fixation—intravital perfusion, postmortem perfusion, and postmortem immersion—was that the membrane should be straight or slightly displaced in one way or the other. Any other position, including complete collapse and rupture with or without compression of the organ of Corti, we considered as artifact.

Mygind[10] found these changes almost exclusively in specimens fixed with intravital perfusion and consequently maintained that they are produced by the combined action of washing out the blood vessels with saline (first phase) and perfusion of fixative (second phase). His working hypothesis is based on the following: 1. The anatomical arrangement of the vascular supply to the vestibular region of the spiral ligament and of the stria vascularis. 2. Physiological considerations. The perilymph is a filtrate which is mostly produced in the vestibular region of the spiral ligament. The endolymph is mostly a secretion of the stria vascularis and consequently depends upon the metabolic state of these cells. 3. Physicochemical considerations, as follows: During perfusion of saline, as colloids are removed, the colloid-osmotic pressure in the capillaries is eliminated; consequently, water filters into the perilymphatic space and produces an increase in pressure. This overpressure is balanced by a concomitant secretion of water into the stria vascularis. If the filtration mechanism predominates, which according to Mygind "is largely due to caprices of a nervous-vascular mechanism," the balance is broken. Reissner's membrane collapses, and the endolymph is pushed out through the canalis reuniens toward the endolymphatic sac. Similar changes take place in the vestibular system. The final displacement of Reissner's membrane occurs only when the balance between the two systems is reestablished. In extreme cases, the membrane collapses over the organ of Corti and stria vascularis, or it is broken and the overpressure is applied on these structures, giving the familiar picture of endolymphatic compression. The second phase takes place during perfusion of fixative. The endolymphatic sac shrinks during fixation, and the endolymph returns toward the cochlear duct and thus elevates Reissner's membrane. This elevation, in turn, pulls the tectorial membrane off the organ of Corti. Mygind claims that the stigma of the first phase can always be traced.

In our series with intravital fixation (26 ears), localized collapse of Reissner's membrane was found in a few cases. In one of them, the collapse reached the organ of Corti without compressing it. In the region of collapse, in each of these cases, the blood

vessels of the stria vascularis and spiral ligament, including the vestibular portion, were partially or totally obliterated with red cells. In other words, the regions were either partially perfused or not perfused at all, which suggests that the changes in the position of the membrane are more related to a poor fixation than to a predominant filtration. The amount of saline for washing out the blood vessels may be a factor, also. However, in our series with intravital fixation, where 50, 100, 200, and 300 cc. of saline were used, no difference in the position of the membrane was found from one specimen to another.

The theory of Mygind assumes that the position of the membrane is due to an imbalance between the filtrating and secreting mechanisms. Thus an alteration of the latter should bring about the picture of collapse of Reissner's membrane quite frequently. The temporal bones of animals exposed to severe metabolic impairment were restudied.[11] These animals received several bouts of fulminating anoxia or asphyxia until depression of cochlear responses for about three hours was obtained. The animals were killed by intravital perfusion. The results showed that Reissner's membrane presented the same variations as found among the controls. Furthermore, the balance between the two mechanisms should be largely altered after death. Postmortem perfusion was carried out in 24 animals, as indicated in the method. The perfusion was successful in those animals dead for less than five hours. Between five and seven hours only small regions of the inner ear were perfused, and after seven hours the procedure was apparently unsuccessful.

In successful postmortem perfusion (20 animals), Reissner's membrane was in standard position, although in a few cases it was partially collapsed over the limbus. These observations suggested that the metabolic state of the cells does not play an important role during perfusion. Therefore the hypothesis of Mygrind regarding imbalance between filtration and secretion of water during perfusion of saline may be questionable. The most prominent feature in the incomplete or unsuccessful postmortem perfusion (eight animals) was either a tendency of Reissner's membrane to bulge (four ears) or that it was broken and collapsed over the stria vascularis and organ of Corti (seven ears). In the remaining five ears, Reissner's membrane was straight (two ears) or displaced toward scala media (three ears). It seems clear that in those cases where Reissner's membrane bulged or was broken, an overproduction of fluid in the scala media took place, and consequently the conditions for compression of the organ of Corti were fulfilled. In Figure 7 a representative case perfused five hours after death, clearly illustrates these findings. The Reissner's membrane was broken (Fig. 7 *b*) and agglutinated over the stria vascularis and organ of Corti. The picture corresponds to the cases reported by Mygind [10] as moderate compression. The overproduction of fluid may be due to pure filtration in the stria vascularis or rupture of some vessels, although no sign of rupture was found in the serial sections. Whatever the mechanism may be, it is reasonable to think that Reissner's membrane is broken because its elastic properties are lost in advanced autolysis. Under these experimental conditions, the findings seem to support the working hypothesis of Mygind,[10] that compression of the organ of Corti is brought about by pressure. But they do not support his view that the phenomenon is intravital because it appeared only when perfusion was made later than four to five hours after death.

Compression of the organ of Corti is an important phenomenon from both theoretical and practical points of view. First of all, there are several degrees of compression, from slight distortion to such complete collapse that no cells can be identified. Mygind [10] and Wittmaack [12] stated that this is an intravital phenomenon, although each one gave a different explanation of its mechanism. The practical point is that we did not identify compression in our series of animals with intravital perfusion, successful postmortem perfusion or postmortem im-

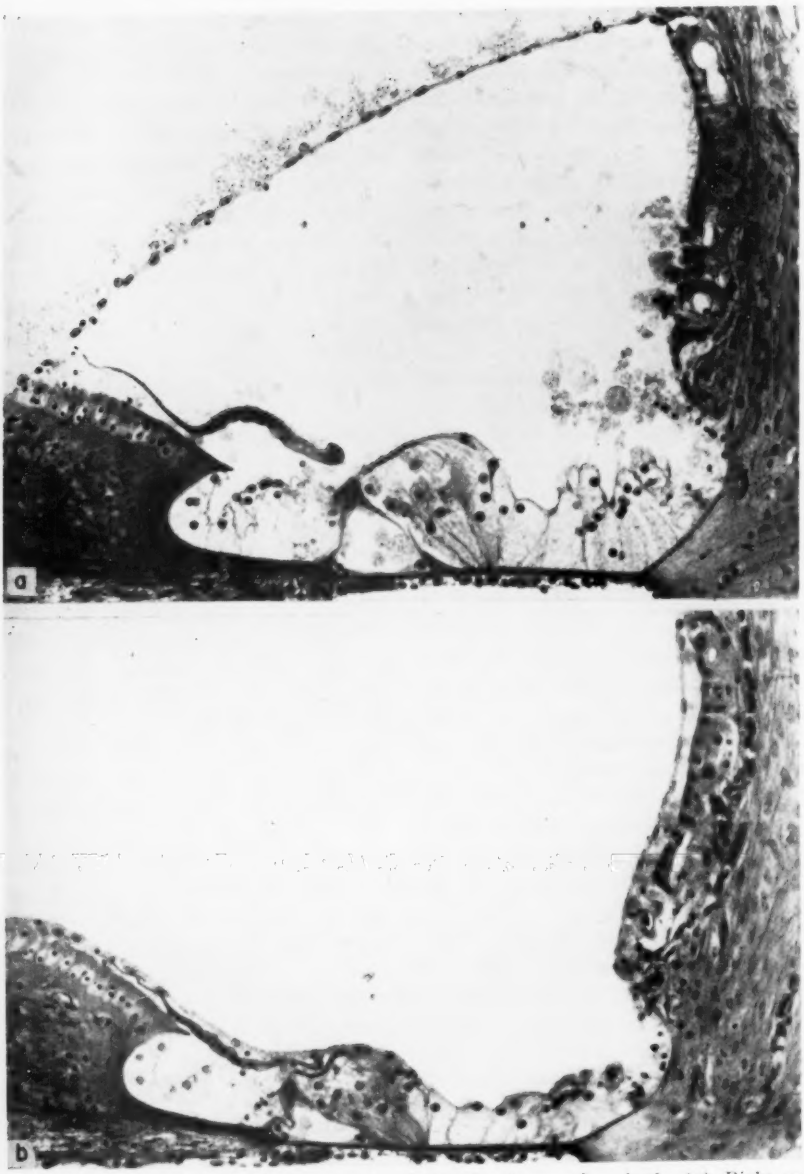

Fig. 7.—Guinea pig B-48. Postmortem perfusion five hours after death. (*a*) Right ear. Reissner's membrane displaced toward scala vestibuli. Advanced autolysis. (*b*) Left ear. Reissner's membrane broken and collapsed over stria vascularis and organ of Corti. Although the autolytic changes are similar to the right ear, they do not appear so prominent because of the compression brought about by the collapse of Reissner's membrane. Both microphotographs × 240; Mallory's azan staining.

mersion; however, it is a common feature of human specimens. As we shall see in a later paper, these observations support an old conviction that compression of the organ of Corti so that no structures can be recognized is an artifact produced by some error in the histological technique.

Mygind's hypothesis regarding the cause of collapse of Reissner's membrane has two implications which are questionable. The first is the displacement of a mass of fluid of viscosity higher than water through a narrow passage, the canalis reuniens. It may be possible that a large perilymphatic pressure could push the endolymph through the canalis reuniens, which may distend, thus facilitating the passage. But the return of the endolymph during perfusion of fixative is obscure because, if the endolymphatic sac shrinks, so do other structures, including the canalis reuniens, and consequently the return should be impeded. Under these circumstances it is reasonable to guess that the endolymph would remain and dilate the vestibular walls. Evidence of this anticipated event was never found.

The second implication of Mygind's hypothesis is that the final position of Reissner's membrane is attained during perfusion of fixative. We think that considerable disturbance of the labyrinthine fluids is produced by further fixation, decalcification, washing, dehydration, and embedding, which may account for the phenomenon. We have no information about the effect of these disturbances upon the position of Reissner's membrane. However, changes in its elastic properties were observed from one step of the histological technique to another. Four guinea pigs were killed by intravital perfusion, and the temporal bones were treated by the routine method. At various stages, a cochlea (a different one at each stage) was dissected under binocular microscope (40 ×). With fine instruments, the otic capsule was removed from apex to base, Reissner's membrane exposed, and the elasticity tested with a fine stylet. Between perfusion and dehydration the membrane was flag-like. During dehydration it became more and more rigid, so that after absolute alcohol it was like a septum which either broke or could be displaced as a whole under pressure. After ether-alcohol it became more brittle, but during embedding with celloidin 3% some rigidity was lost. We realize that these observations are limited because the measurements were rather crude. Nevertheless, they suggested that the final position of Reissner's membrane seems to be attained after dehydration and/or embedding. Whether the position of the membrane can be modified during the interval when it is flag-like as well as during any other step is a question which has not yet been answered. We are looking at the end-result, and from this we cannot evaluate the disturbances which take place from the very beginning of the procedure to the end. Other methods of observation may help to settle this long controversy.

D. Postmortem Changes.—When a cell dies and is not preserved by a fixative it becomes acid, and consequently the enzymes start to work in the wrong direction. The proteins are split into amino acids which diffuse out of the cells. Swelling, homogenization, pyknosis, etc., take place until the cell has disintegrated. The rate of progress of the autolysis depends on many factors, such as temperature, humidity, infection, and metabolic requirements. The physicochemical reactions of the fixative and other reagents with the compound of the cell in autolysis must be different at any step, since the chemical composition of the cell changes continuously as autolysis progresses. Thus, the microscopic picture will depend upon time elapsed between death and fixation, fixative and staining, autolytic stage, etc. Histochemical methods are the most appropriate to substantiate these changes. Routine methods such as hematoxylin-eosin or Mallory's azan staining and light microscopy give no information about the nature of the phenomenon.

The autolytic changes in the inner ear usually followed a characteristic pattern. Swelling and homogenization of the cells appeared first, followed by precipitates and

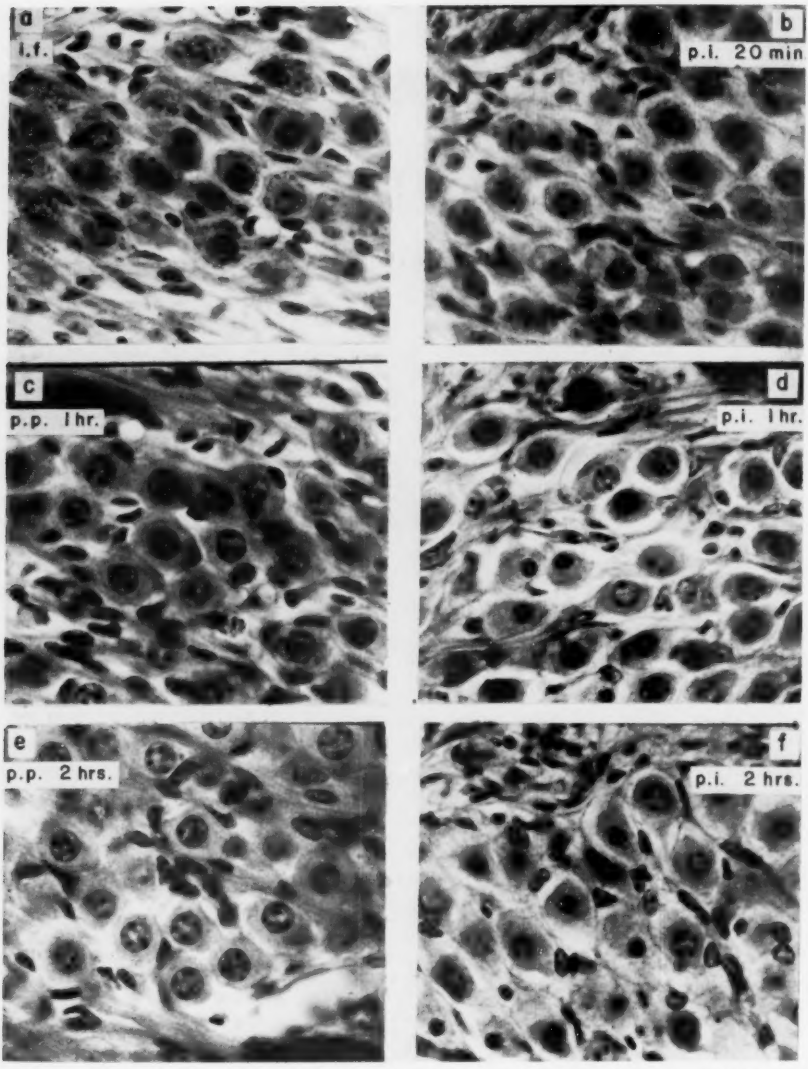

Fig. 8.—Postmortem changes in the ganglion of Corti. Comparison between perfusion and immersion procedures. In this and subsequent figures the following symbols are used: i. f., intravital fixation; p. p., postmortem perfusion; p. i., postmortem immersion. Also, time between death and perfusion or immersion is indicated on each photomicrograph. (*a*) Intravital fixation. (*b*) Immersion two minutes after decapitation. Tigroid substance is absent, and constituents of the nuclei are not well differentiated. In (*c*) and (*e*) swelling of cells is present and tigroid substance lost, but constituents of the nuclei are preserved. In (*d*) and (*f*) swelling, homogenization, and pyknosis are predominent. Hematoxylin-eosin staining; × 480.

globular formation, pyknosis, cellular detachment, and, finally, disintegration. However, a significant difference in the autolytic changes was found as the two methods of fixation, postmortem perfusion and postmortem immersion, were compared. For a better understanding each structure will be described separately and an attempt to illustrate the most important changes will be made.

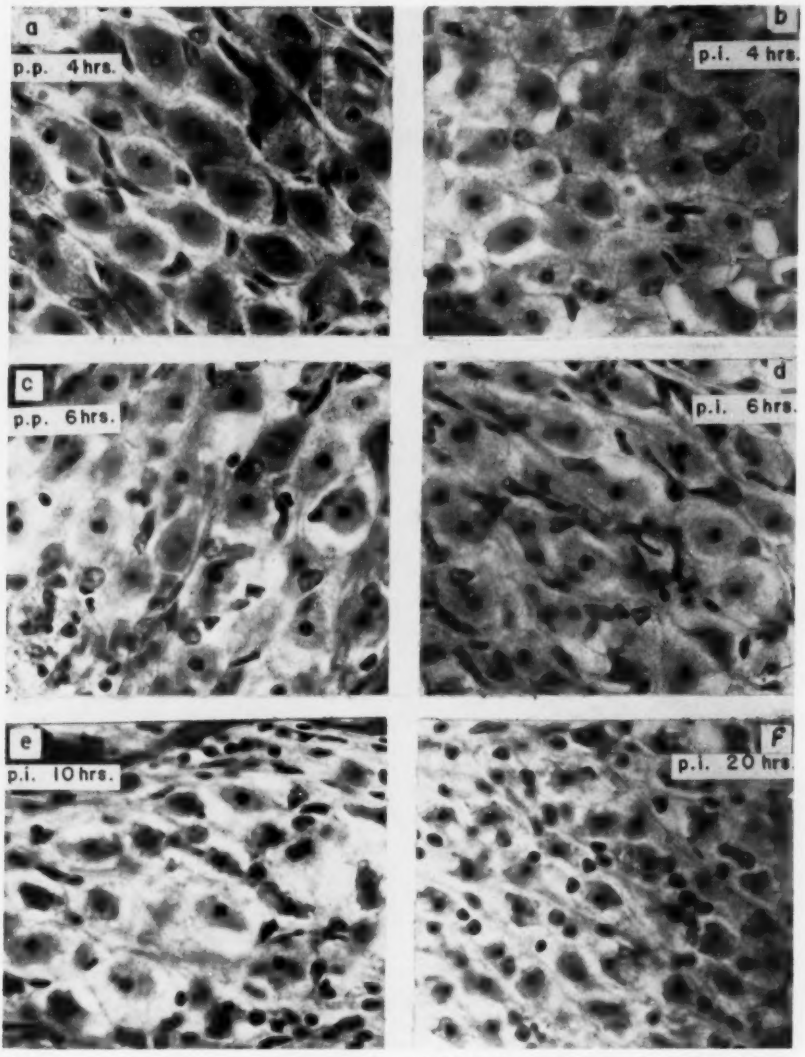

Figure 9.—Postmortem changes in ganglion of Corti (later stages). Comparison between perfusion and immersion procedures. In (*a*) about 60% of ganglion cells are pyknotic, while in (*b*) 100% are pyknotic. In (*c*) perfusion was incomplete and the results are about the same as in (*d*). In (*e*) ganglion cells are in disintegration, and in (*f*) disintegration is almost complete but pyknotic nuclei remained. Hematoxylin-eosin staining; × 480.

Ganglion of Corti: The first noticeable change was swelling and homogenization of the cell bodies, which appeared in postmortem immersion specimens no matter how fast the temporal bones were dissected and immersed in fixative. Figure 8 *b* illustrates the cell bodies of the ganglion of Corti of one animal in which time for decapitation, dissection of temporal bones, and immersion in fixative was two minutes. Swelling and homogenization were already present. Pyknosis appeared approximately one hour after death (Fig. 8 *d*) and in later stages (7-10 hours) only the cell borders and some homogeneous precipitate around the pyknotic nuclei could be identi-

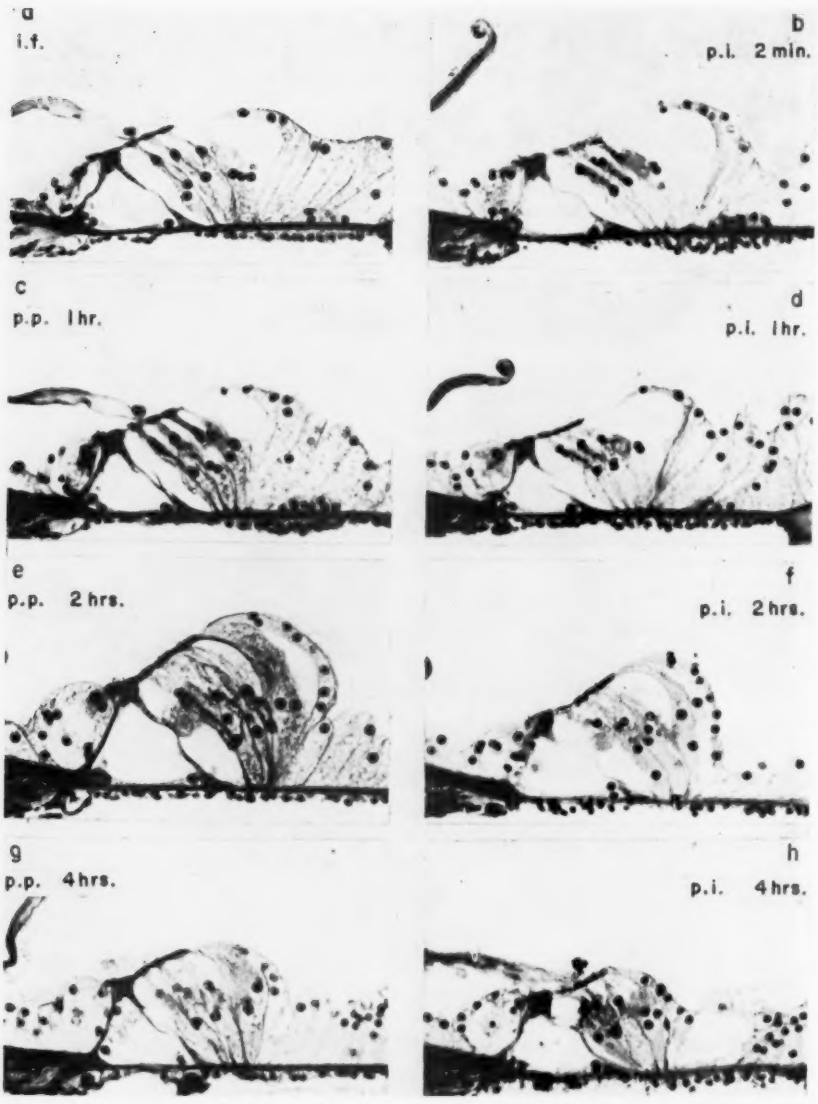

Fig. 10.—Postmortem changes in the organ of Corti (early stages). Comparison between perfusion and immersion procedures. (*a*) Intravital fixation; normal cytoarchitecture. (*b*) Immersion two minutes after decapitation. Homegenization and loss of border cells is the most prominent feature. In (*c*), (*e*), and (*g*) swelling and some precipitates appeared but nuclei, cell borders, and granular-like cytoplasm are preserved, while in (*d*), (*f*), and (*h*) the autolytic changes are more prominent and disintegration of some cells has started. Mallory's azan staining. This stain was used for a better differentiation of precipitates, cell borders, and granular-like cytoplasm. The nuclei are not as well differentiated as with hematoxylin-eosin. All photomicrographs × 240.

fied (Fig. 9 *e*). Finally, at about 20 hours, all structures disintegrated, but the pyknotic nuclei remained (Fig. 9 *f*).

In the specimens fixed by postmortem perfusion, homogenization appeared after about 20 minutes and pyknosis after two

hours (Fig. 9 *a*). As far as perfusion was complete, the increase in pyknosis was moderate. In incomplete perfusion the autolytic changes become more similar to those in postmortem immersion; no difference was found between the two methods when perfusion was unsuccessful. The observations also seem to indicate that the rate of progress of autolysis of nerve elements was as fast as that of elements of the central nervous system and faster than any other structures of the inner ear. The high metabolic rate of nervous tissue may account for the difference, but the question will remain until more rational methods of investigation are used.

In the ganglion of Corti, as well as in the other structures of the organ of Corti, the cell population is better preserved by successful postmortem perfusion than by postmortem immersion. The explanation of the difference is rather simple. If intravital fixation or postmortem perfusion is used, the tissue is readily fixed. If postmortem immersion is used, the rate of fixation depends mostly on temperature, size of the sample, type of tissue, and penetrating coefficient of the fixative. We shall refer to the effect of temperature in a later chapter. The specimens used in this study were quite large; that is, a block of about $10 \times 18 \times 22$ mm., formed mostly of bone tissue. The fixative was Heidenhain-Susa, in which mercuric chloride and formaldehyde are the fixative compounds. According to the data of Baker,[7] these have a relatively high penetrating coefficient in liver, spleen, and kidney. But the inner ear structures are almost completely surrounded by bone. Consequently, the penetration of the fixative is largely impeded. The only pathways by which it may reach the inner ear structures are narrow passages, such as the internal meatus (filled with nervous tissue), the cochlear aqueduct, the oval window (occupied by the footplate of the stapes) and the round window (closed by a fibrous membrane). Thus, penetration of the fixative would be slow, and there is a long interval (probably hours) between immersion of the temporal bone and fixation of the inner ear structures. During the interval between immersion of the samples and fixation of the structures, the autolytic process progresses steadily; so the changes observed in temporal bones fixed by postmortem immersion are not those present at the moment the specimen is immersed in the fixative, but include those which take place after the immersion.

Ganglion of Scarpa: The elements of this ganglion followed the same autolytic changes as the ganglion of Corti for postmortem perfusion. In postmortem immersion, however, Scarpa's ganglion was better preserved. The explanation probably has to do with the anatomical arrangement. Scarpa's ganglion is in the inner meatus and therefore would be reached by the fixative sooner than the ganglion of Corti, almost completely surrounded by bone in Rosenthal's canal, could be reached.

Organ of Corti: Swelling and homogenization of cells, detachment of Hensen's from Deiters' cells, space formation among external and internal sulcus cells, and some precipitates may be found in specimens immersed in fixative two minutes after decapitation. As the interval between death and immersion was prolonged these changes became more pronounced. Between one and two hours, pyknosis and detachment of internal sulcus and internal hair cells were initiated. The surface became irregular, the granular-like aspect of cells was lost, the amount of precipitate increased, and globular formation may be seen in some spots. Thereafter disintegration of the organ of Corti progressed for many hours. Variations among individuals as well as among regions of the same cochlea, were often found. In general, internal hair cells and neighboring internal sulcus cells were the first to disintegrate. The inner pillars of Corti were detached and pushed, together with some of those cells, into the tunnel of Corti. Later, disintegration of the external sulcus cells may take place, or the core of the organ of Corti, formed by external hair cells, external pillars, Deiters'

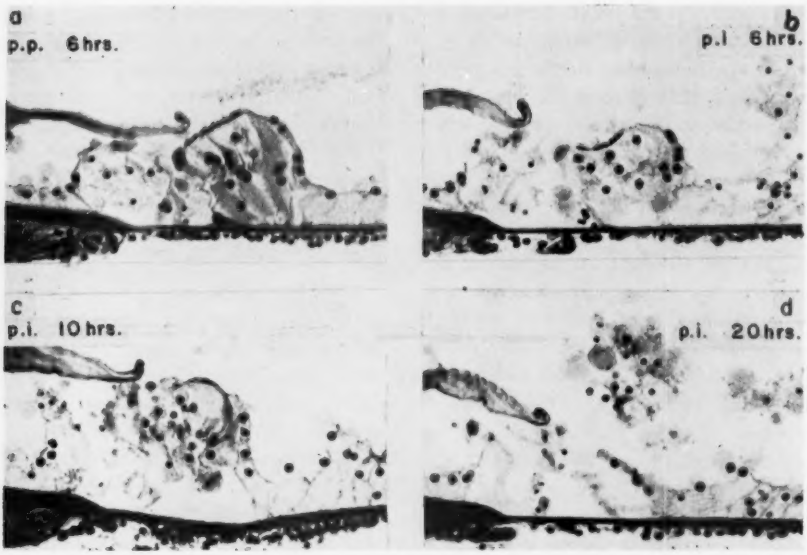

Fig. 11.—Postmortem changes in the organ of Corti (later stages). (*a*) Perfusion failed and autolysis appeared as advanced as in (*b*). In (*c*) the core of the organ of Corti was detached from basilar membrane. In (*d*) only a few cells remain; the others are disintegrated in scala media where the pyknotic nuclei remain imbedded in precipitates and globular formation. Mallory's azan staining; × 240.

and Hensen's cells were detached and dissolved. In Figures 10 and 11 an attempt is made to illustrate these changes as a function of time (from two minutes to 20 hours).

The specimens fixed by postmortem perfusion showed first a small but progressive swelling of the cell population. Later, between two and four hours, some precipitates and globular formation appeared. Aside from these changes the microscopic cytoarchitecture was well preserved. As soon as the perfusion was unsuccessful, the same result was obtained by this method as by immersion. In Figures 10 and 11 the autolytic changes after postmortem perfusion are presented for comparison with postmortem immersion. In the former the most prominent feature is the cellular edema, but nuclei, cell borders, and granular-like cytoplasm were preserved for four hours. Since the structures were fixed at the time of perfusion, the changes are a good index of the progress of autolysis. When the actual observation under the microscope is made, the preservation of all structures of the inner ear is remarkable for about four hours or until perfusion fails. This suggests that the autolytic process does not progress faster in the inner ear structures than in other tissue, as Nager and Yoshii [1] claimed. Unfortunately, the method of postmortem perfusion not only fails four to five hours after death, but introduces some artifacts, overproduction of fluid, as was illustrated in Figure 7. Our knowledge of the sequence of changes which take place more than four or five hours after death is based on a misleading procedure, postmortem immersion. The attempt to improve this method by removing the stapes was a failure, because not only were the same results obtained, but they were complicated by artifacts, collapse of Reissner's membrane and vestibular walls.

Tectorial Membrane: Detachment of the tectorial membrane from the organ of Corti was a common feature found in the two procedures. Detachment from the limbus appeared in postmortem immersion specimens, four hours after death, and in

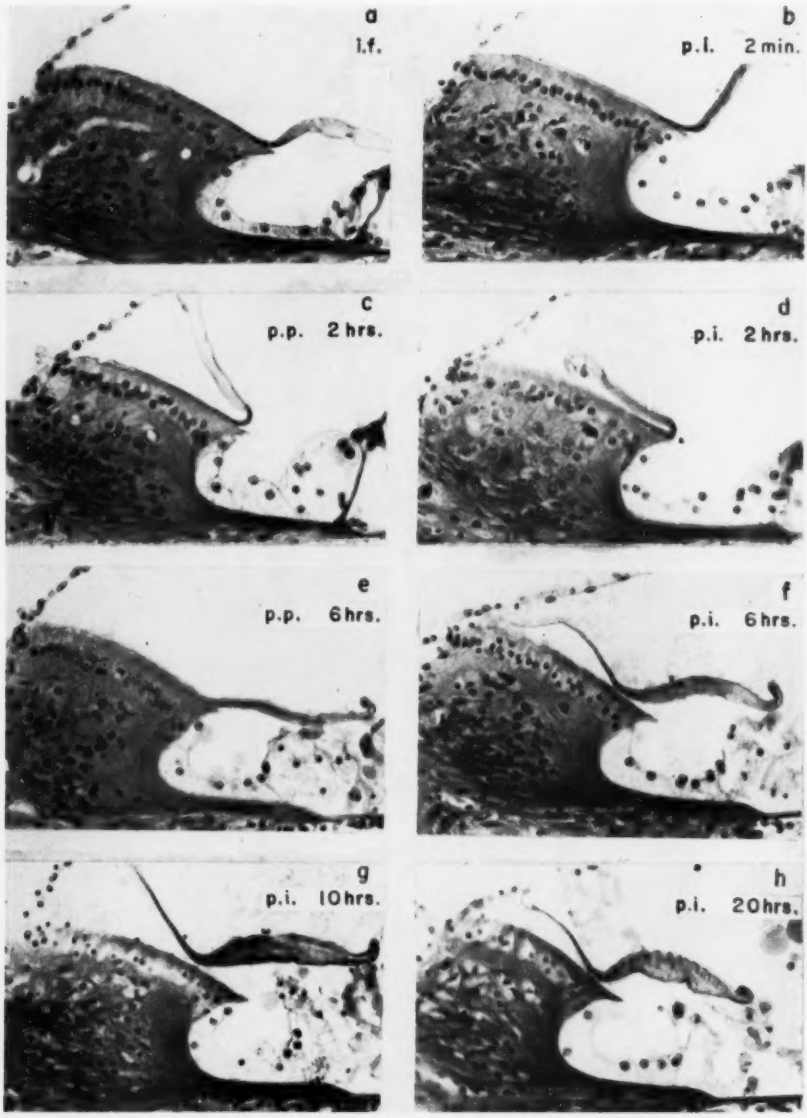

Fig. 12.—Postmortem changes in the limbus. Comparison between the two procedures of fixation showed preservation of cells of interdental groove until perfusion failed. In immersion procedure pyknosis started in early stages, at about one hour. Notice that tectorial membrane is detached from the limbus but did not disintegrate. Mallory's azan staining; × 240.

postmortem perfusion when it was unsuccessful.

The tectorial membrane may swell a little, but no signs of disintegration of this structure were found even at 20 hours after death (Fig. 12).

Limbus: Pyknosis of the cells of the interdental groove appeared one to two hours after death (postmortem immersion). Between 7 and 10 hours the cells became detached from the grooves, and at 20 hours only a few remained. No changes in the

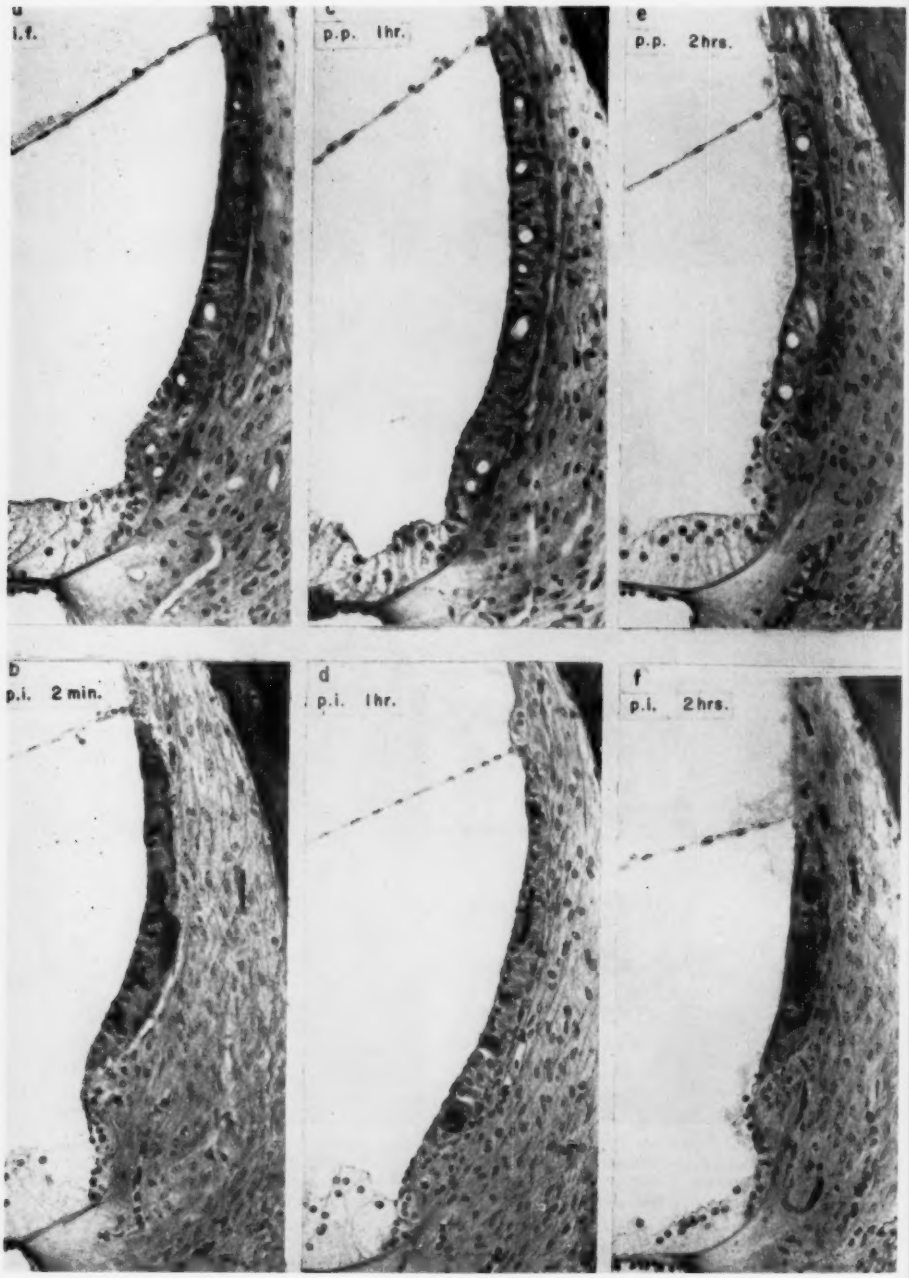

Fig. 13.—Postmortem changes in the spiral ligament and the stria vascularis (early stages). At two hours after death, pyknosis and detachment of cells of the spiral prominence appeared in specimens perfused as well as in those which were immersed. The cell constituents of the spiral ligament are well preserved with either method of fixation. In specimens perfused, globular formation in the stria vascularis and edema of its cells appeared about one hour after death and increased thereafter. In the specimens immersed, homogenization of cells of stria vascularis and some precipitates are present. Mallory's azan staining; × 240.

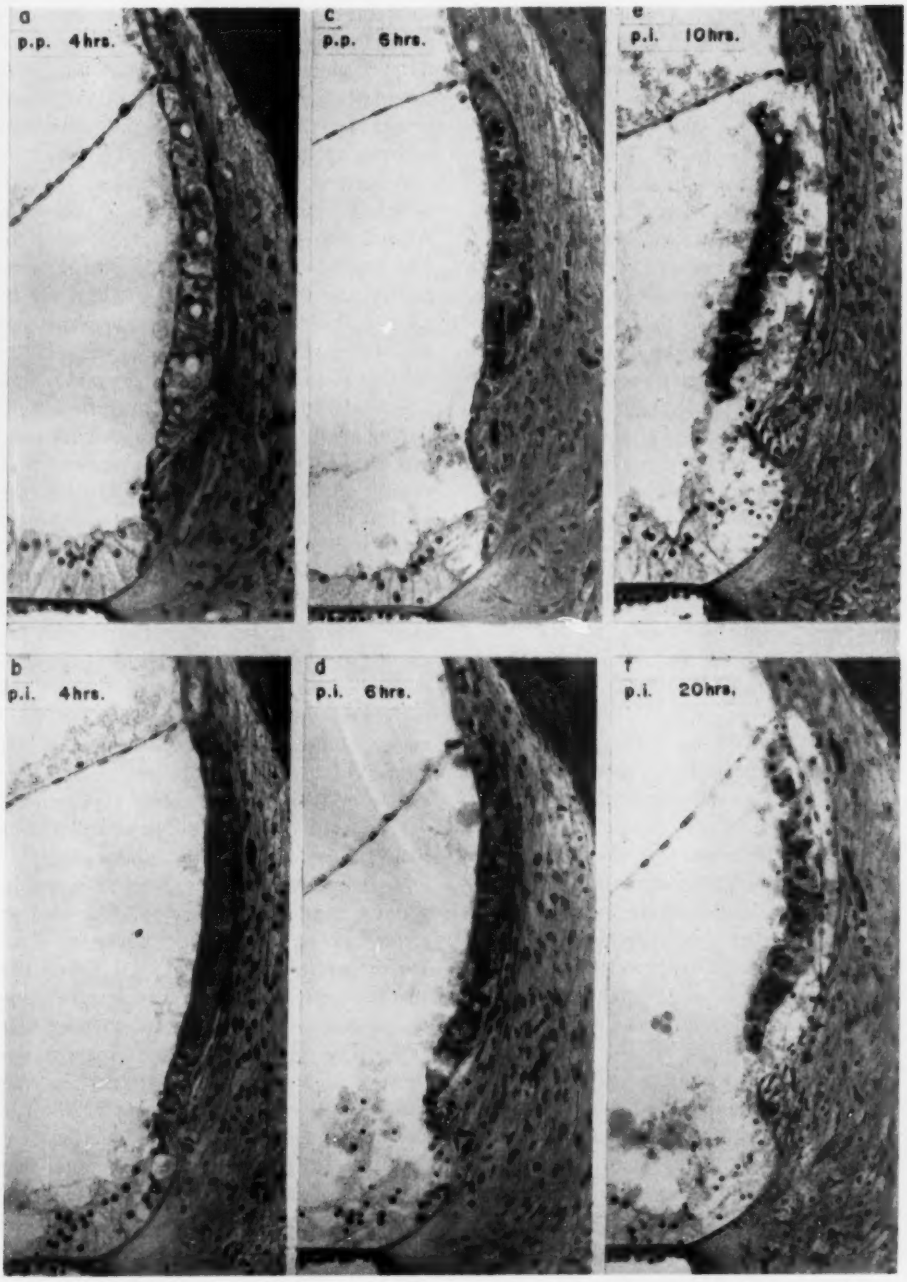

Fig. 14.—Postmortem changes in the spiral ligament and the stria vascularis (later stages). In (*a*) edema of the stria vascularis and accumulation of globules on its surface are very prominent, while in (*b*) they are not present. In (*c*) perfusion failed, and edema and globules disappeared. The result was the same as in (*d*) (immersion). In later stages the stria vascularis was detached and fragmented. Notice that cells of spiral ligament are rather well preserved throughout. Mallory's azan staining; × 240.

cells of the interdental groove were found when postmortem perfusion was completed (Fig. 12), and no difference existed between the two methods when perfusion was unsuccessful.

Reissner's Membrane: Postmortem immersion (from two minutes to 20 hours) showed that the position of Reissner's membrane was in most cases straight, but in a few cases it was concave or convex. Postmortem perfusion gave the same results until it failed, in which case the membrane usually was broken and collapsed or displaced toward the scalar vestibuli. Pyknosis appeared in the early stages (one to two hours) with postmortem immersion, while in postmortem perfusion, it was not present until failure of perfusion.

Spiral Ligament and Stria Vascularis: The cells of the spiral prominence were among the first to become pyknotic and detached, as both procedures of fixation demonstrated. The structures of the spiral ligament were preserved until later stages of autolysis (beyond five hours), where pyknosis was the most prominent feature (Figs. 13 and 14). In all specimens fixed by either of the two procedures, precipitates and globular formation were found over the surface of the vestibular region of the spiral ligament. These features also may appear in specimens fixed by intravital perfusion.

Autolytic changes in the stria vascularis after postmortem immersion followed a consistent pattern. Homogenization, precipitates, and sometimes globular formation on the surface, pyknosis, detachment of cells, and finally, between 10 and 20 hours, the stria vascularis was detached and, in some cases, fragmented. The specimens which were well perfused showed pronounced swelling of cells and a characteristic globular formation over the surface. The latter could be observed at about one hour, and as time between death and perfusion was prolonged, their number and size increased. In these specimens the amount of precipitate was negligible until the perfusion was unsuccessful. In Figures 13 and 14 an attempt is made to illustrate the sequence of autolytic changes in the stria vascularis as given by the two procedures. The characteristic globular formation on the stria vascularis after postmortem perfusion brings up the problem of the difference between agonal and postmortem changes.

According to Wittmaack and Laurowitsch [13] they are different processes. Agonal changes are the homogeneous globules deposited on or in the organ of Corti and on the stria vascularis, the swelling of ganglion cells with loss of tigroid substance and some changes in the myelin sheath. According to these authors, the agonal changes are produced by irritation of certain cells during agony and therefore represent a vital process. Subsequent changes appear as a result of autolysis. Falbe-Hansen [8] described the agonal and postmortem changes extensively. He claims that both kind of changes are different stages of the same process, that is, autolysis. Similarly, Mygind [14] found no fundamental difference between agonal and postmortem changes. According to Mygind, agonal changes are not an active process as described by Wittmaack, but represent a decrease in the vitality of the cells and consequently an increase in their water-binding properties.

Our results support the theory that there is no fundamental difference between agonal and postmortem changes, because in our intravital fixation series we failed to demonstrate the agonal changes, even in cases where perfusion was not ideal. It is true that the tigroid substance may disappear, but this change is always related to poor fixation. The agonal changes found in postmortem immersion procedure must be ruled out because—as explained before—the procedure is misleading. Now, if the theory of Wittmaack and Laurowitsch is true, then, assuming the cells survive for some time, the changes should be more prominent immediately after death. The series with postmortem perfusion failed to demonstrate globular formation within the first hour. Furthermore, the ganglion cells retained their tigroid substance for at least 20 minutes after death. Their subsequent ho-

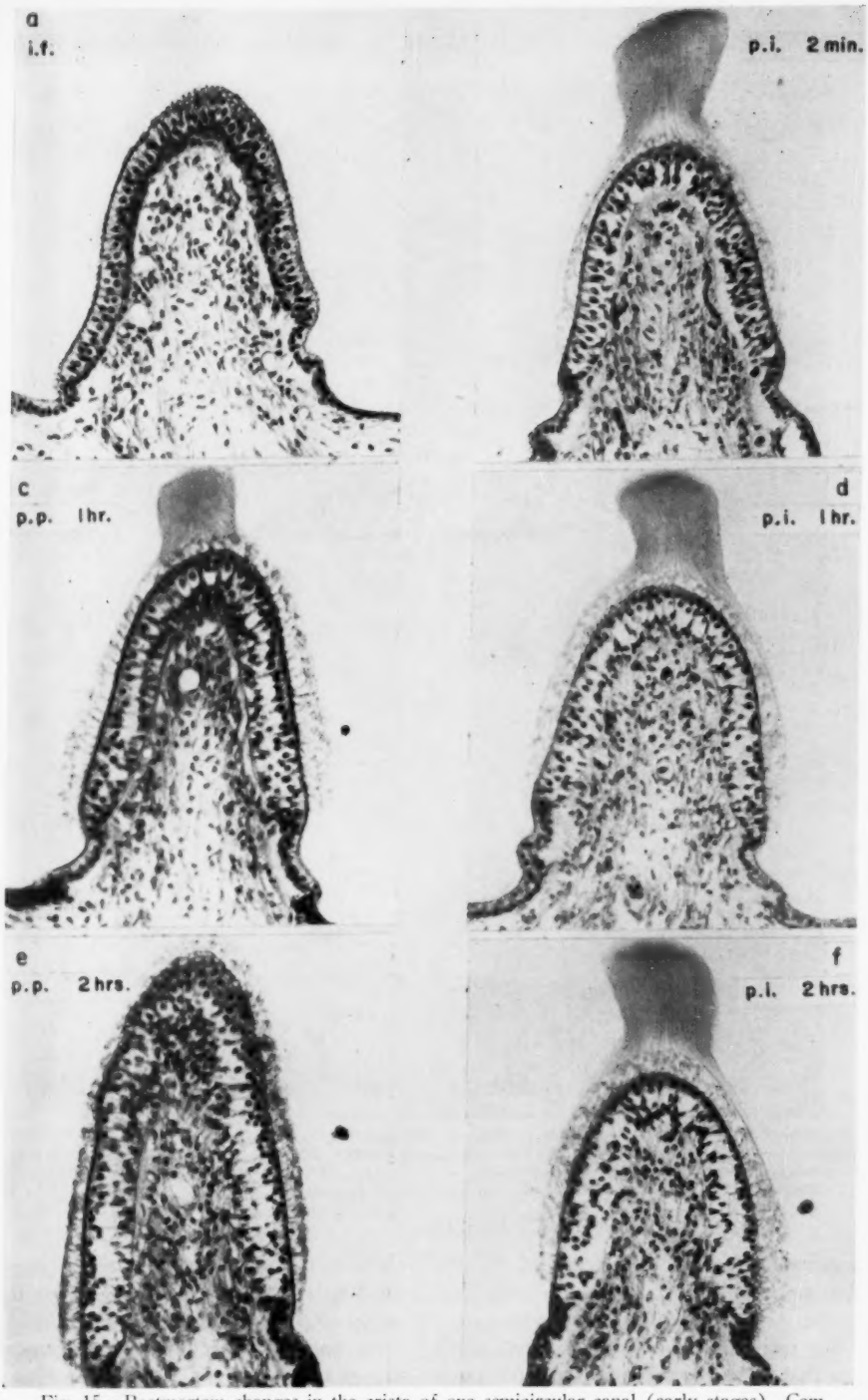

Fig. 15.—Postmortem changes in the crista of one semicircular canal (early stages). Comparison between perfusion and immersion procedures. (*a*) Intravital fixation; normal cytoarchitecture. (*b*) Immersion two minutes after decapitation. Edema of epithelium and precipitates on its surface are already present. In (*c*) and (*e*) two successive stages of perfused specimens showing increase in edema, precipitates, and pyknosis. In (*d*) and (*f*) are the corresponding immersed specimens where edema and precipitates are correspondingly more pronounced. Mallory's azan staining; × 172.

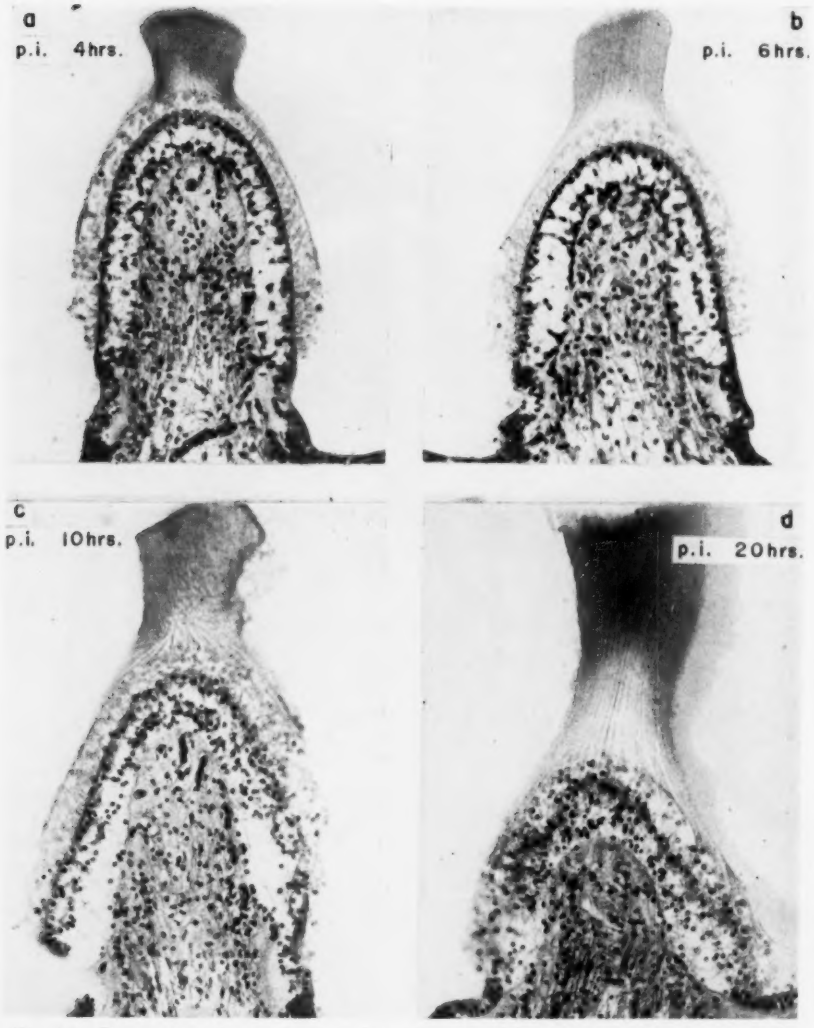

Fig. 16.—Postmortem changes in the crista of one semicircular canal (later stages). Immersion procedure. In (*a*) large amount of precipitates, edema, and some cells are detached as shown by the nuclei found in the cupula. In later stages, the epithelium is detached and finally disintegrates. Notice that the cupular substance, like the tectorial membrane, does not change even at 20 hours after death. Mallory's azan staining; × 172.

mogenization should be regarded as the first sign of autolysis rather than vital changes produced during or after agony.

The increasing globular formation which appeared between one hour and the point at which postmortem perfusion failed, is probably a primary artifact. In support of this hypothesis are the following facts: The characteristic globules are present only on the stria vascularis and some may be found in the vestibular region of the spiral ligament; there is a parallelism between increasing globular formation and edema of the stria vascularis, as Figures 13 and 14 clearly demonstrate, and finally, there is a sudden disappearance of both globules and edema

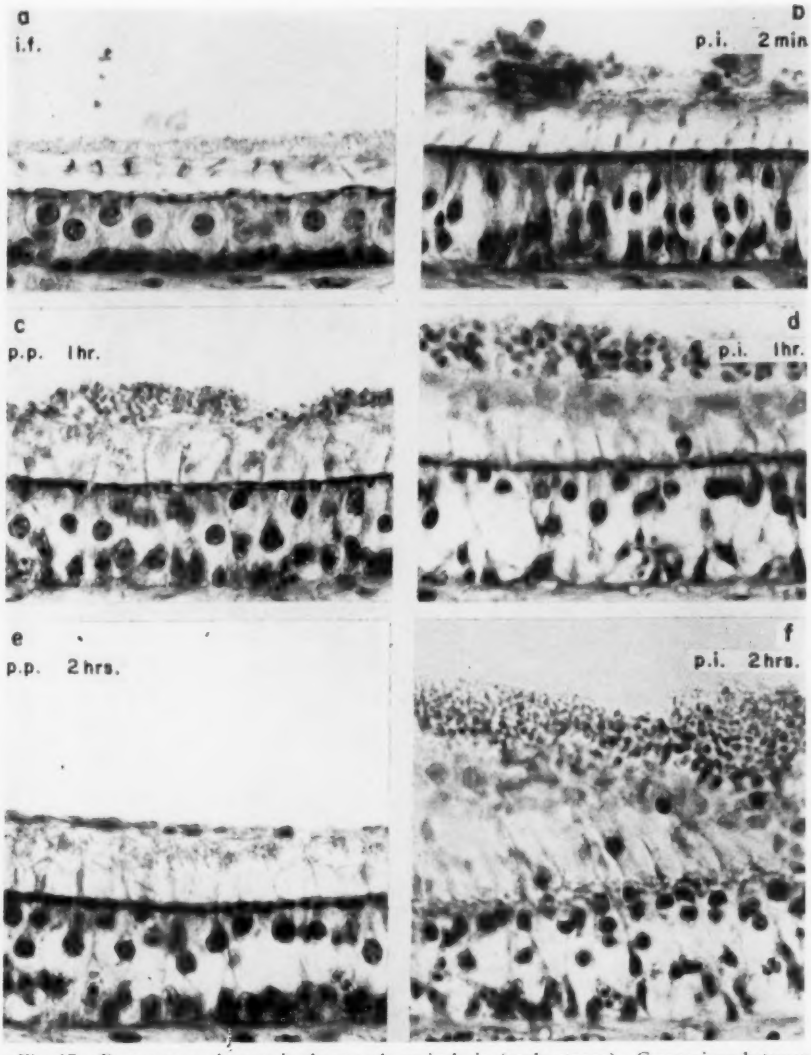

Fig. 17.—Postmortem changes in the macula utricularis (early stages). Comparison between perfusion and immersion procedures. The preservation in the perfused specimens is evident. Notice in (*d*) that one pyknotic cell is just thrown out of the epithelium, and in (*f*) the increase in detached cells. Also notice accumulation of precipitates between otoconia and epithelium and that it is greater in immersed than in perfused specimens. Mallory's azan staining; × 550. (In all photomicrographs only a small region of macula utricularis is shown.)

as soon as the postmortem perfusion fails. These findings all suggest that there is an increase in the water-binding properties of the cells of the stria vascularis in advanced stages of autolysis. The globules probably are a mixture of cytoplasm and water protruding from the cells. Although it seems reasonable to attribute this globular formation to an artifact characteristic of the method, the possibility that they are actual postmortem changes is not completely ruled out.

Vestibular Receptors: The autolytic changes in the vestibular receptors for both

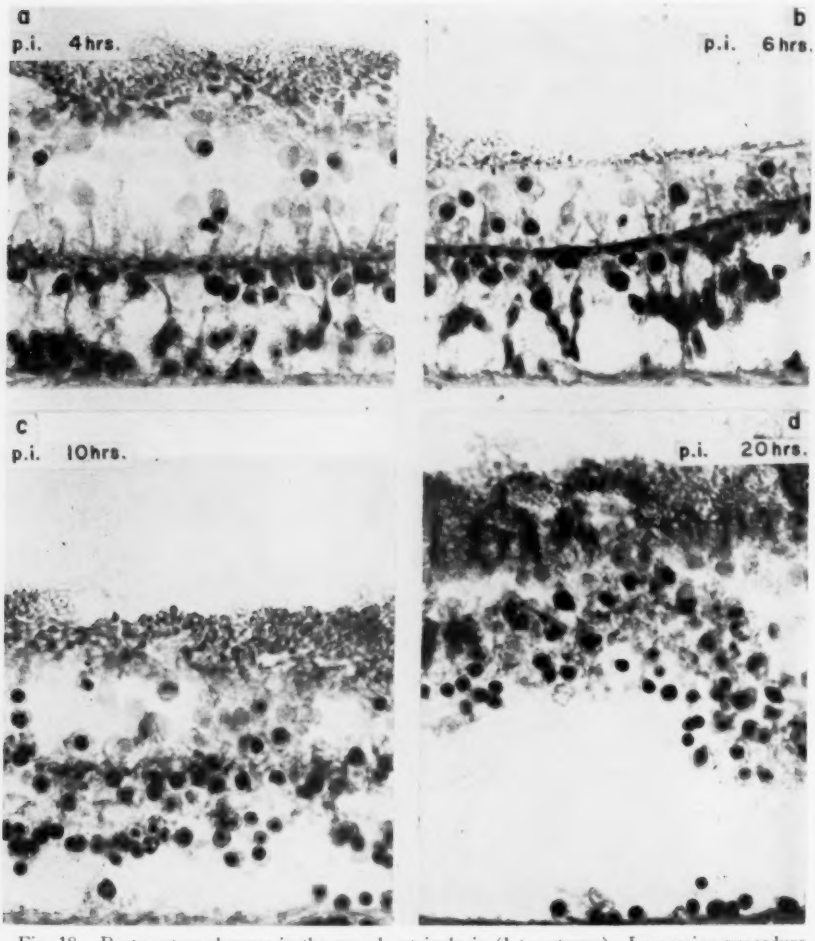

Fig. 18.—Postmortem changes in the macula utricularis (later stages). Immersion procedure. The number of detached cells increased as time between death and immersion was prolonged. At six hours, detachment of epithelium appeared, and finally, at 20 hours, it disintegrated, except for the pyknotic nuclei, which remained. Notice that no significant changes appeared in otoconia. Mallory's azan staining; × 550.

postmortem immersion and postmortem perfusion procedures followed the same general pattern as that described for the cochlear receptors; thus a description is omitted. Figures 15 and 16 illustrate the changes in the crista of a semicircular canal as observed after postmortem perfusion and postmortem immersion. Figures 17 and 18 illustrate the changes in macula utricularis as observed in the two methods.

The autolytic changes in the vestibular as well as the cochlear receptors of the set of animals killed by decapitation or asphyxia were the same. In the group of animals with stapes removed the macula saccularis and sometimes the macula utricularis showed a better preservation than other structures. Again this set of animals demonstrated that penetration of fixative into the inner ear is quite a slow process.

Effect of Temperature: It is well known that the rate of progress of autolysis diminishes as temperature is lowered. The temporal bones of the animal kept in re-

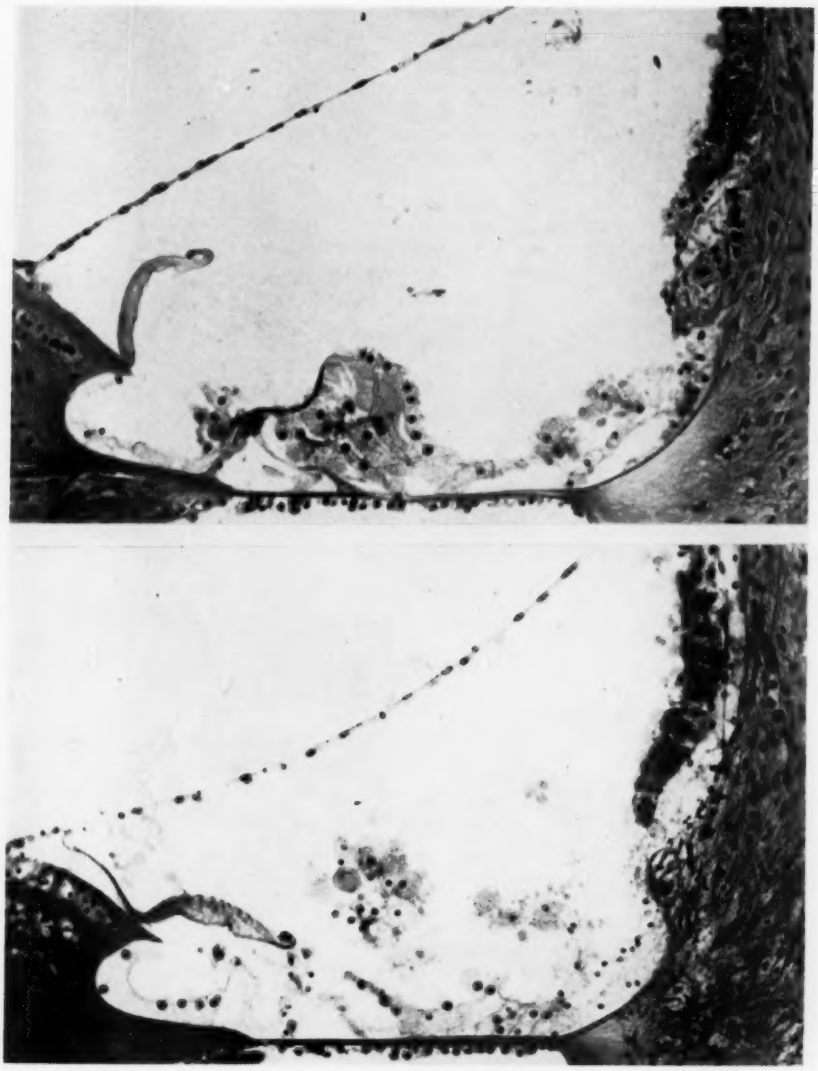

Fig. 19.—Effect of temperature upon postmortem changes. (Upper) Organ of Corti of one animal kept under refrigeration (4 C) for 20 hours after death. There are advanced postmortem changes, but the structures can still be recognized. (Lower) Organ of Corti of one animal, kept at room temperature (about 25.5 C) for 20 hours, was completely disintegrated. Mallory's azan staining; × 240.

frigeration (4 C) for 20 hours confirmed these general findings. In Figures 19 and 20 a comparison is made with a specimen which remained at room temperature for the same length of time. The figures show that in the refrigerated animal the structures are better preserved.

Summary and Conclusions

A study was made under light microscopy of the sequence of histologic changes of the vestibular and cochlear receptors in 82 guinea pigs as autolysis progressed.

Intravital fixation with Heidenhain-Susa solution and staining with hematoxylin-eosin

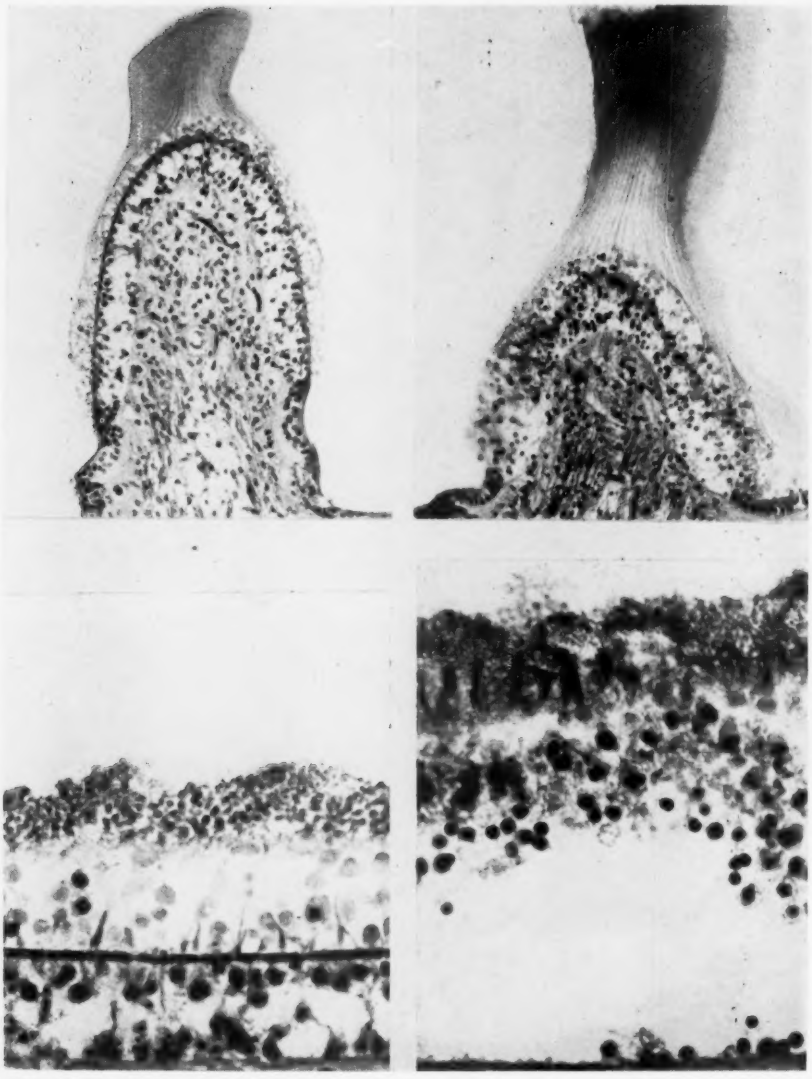

Fig. 20.—Effect of temperature upon crista of one semicircular canal and macula utricularis. The left column shows the structures of one animal kept under refrigeration (4 C) for 20 hours. The right column illustrates the corresponding structures of one animal kept at room temperature (about 25.5 C) for the same length of time. The difference in postmortem changes between the two specimens is evident. Mallory's azan staining; crista, × 172; macula utricularis, × 550.

emphasized the nuclear structures of the cell population of the inner ear. The cytoplasmic constituents were absent, but the tigroid substance of ganglion cells remained. This result was called the normal microscopic cytoarchitecture of the inner ear. Each method of fixation and staining has its own characteristics, and consequently the standard must be defined for each procedure. Any nonpathologic changes which may appear in the normal microscopic cytoarchitecture were considered to be artifacts. The

commonest were detachment of the tectorial membrane from the organ of Corti, detachment of Hensen's from Deiters' cells, space formation in internal sulcus and/or external sulcus cells, and collapse of Reissner's membrane with or without compression of the organ of Corti.

With postmortem perfusion, the first changes (loss of tigroid substance and swelling) appeared in the ganglion cells approximately 20 minutes after death. Swelling, homogenization, and pyknosis progressed rapidly in the nervous elements. In the nonnervous tissue, the autolytic process was slower, the first sign appearing as swelling of the cells of the cochlear and vestibular receptor, approximately one hour after death.

In postmortem immersion, penetration of the fixative was a slow process, taking probably hours to reach the inner ear structures, and therefore the changes which appeared were nothing other than postmortem changes.

The study pointed out that agonal changes (formation of globules and loss of tigroid substance) are features of the autolytic process. Also, the rate of progress of postmortem changes in the inner ear structures seems to be about the same as that of other tissues.

Localized atrophy of the stria vascularis, with or without degeneration of the organ of Corti, was found in some young, healthy guinea pigs. These lesions suggest the possibility of vascular accident.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Transtympanic Mobilization of the Stapes

Effects on the Transmission Characteristics of the Middle Ear

FRANK KODMAN Jr., Ph.D., and GERALD SPERRAZZO, M.A., Lexington, Ky.

Transtympanic mobilization of the stapes was initiated approximately 70 years ago and revived more recently by Rosen.[4] When the otological and audiological results indicate that the stapes has been "ankylosed," the otologic surgeon enters the middle ear via the tympanic membrane after administering a local anesthetic. The surgery is usually performed through an ear speculum with use of optical magnification and an instrumental mobilizer to improve the transmission of sound by the ossicles.

Although stapes mobilization is used primarily to improve impaired hearing, the procedure is analogous to an experimental manipulation of the ossicular chain. Allowing for etiological and pathological variations of middle-ear disease, there results a sampling of these processes as the number of surgical cases increases, since no two cases are exactly identical. Thus an analysis of a sample of pre- and postsurgical pure-tone thresholds should reveal the resultant differential effects attributable to the transmission characteristics of the middle ear.

Forty-four surgical cases were selected equally from two surgeons, Juers[3] and Scheer.[5] Each case showed significant post-

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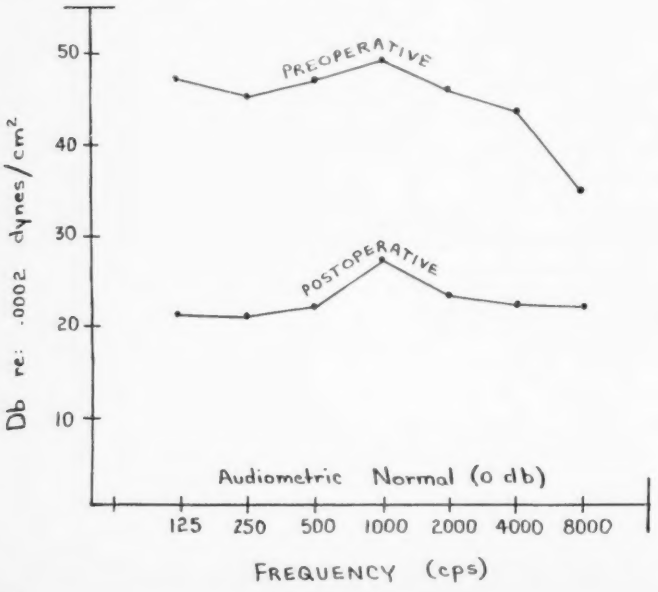

Mean thresholds, pre- and postoperative, for octave frequencies from 125 to 8000 cycles.

operative improvement. Audiometric measurements (air-conduction thresholds) were made before and after stapes mobilization at the octave frequencies 125, 250, 500, 1000, 2000, 4000, and 8000 cycles. In the Figure are shown the mean thresholds, pre- and postoperative, for each frequency. The reader will note that there is a greater loss in the low tones of the preoperative thresholds, while the postoperative trend is toward an over-all flat type air-conduction loss except at 1000 cycles.

Preoperative Thresholds

Before beginning a statistical analysis of the effects of stapes manipulation, it is important to examine the thresholds of the sample prior to the surgical procedures. When the raw differences between the preoperative thresholds were subjected to statistical test, a significant result was found. χ^2 was 130.57 and is significant beyond the 0.01 level, indicating that we can reject the hypothesis that the preoperative threshold differences are due to chance.[2] In other words, our sample showed significantly different preoperative thresholds at the seven frequencies considered.

Preoperative vs. Postoperative Thresholds

In selecting our sample, we were interested primarily in cases which showed successful postoperative improvement. The differences between the mean preoperative and the mean postoperative thresholds resulted in a χ^2 significant beyond the 0.01 level. χ^2 was 7.28. We can accept the differences between the preoperative and postoperative thresholds with a high degree of confidence. On this basis, we can consider our sample as constituting only postsurgical cases showing significant improvement.

Postoperative Thresholds

A χ^2 of 53.09 was found between the postoperative frequencies, indicating that they differed significantly from each other. The greatest difference was contributed by 1000 cycles. Thus our sample is characterized by different preoperative thresholds, different postoperative thresholds, and a significant difference between the preoperative and postoperative results.

Lower Frequencies vs. Higher Frequencies

An important consideration is whether or not the lower frequencies (125, 250, and 500 cycles) improved more than the higher frequencies (2000, 4000, and 8000 cycles). A χ^2 of 168.2, significant beyond the 0.01 level, was obtained. In terms of gross decibel gain, the gross differences favored the lower frequencies, indicating that they improved significantly more than the higher frequencies. The largest numerical difference occurred between the terminal frequencies 125 and 8000 cycles. Bone-conduction measurements were not available on the entire sample; therefore we were unable to equate the cases for inner ear pathology. A mixed loss was present in some of the cases.

Next, the percentage of improvement at each frequency was considered. For the lower frequencies (125, 250, and 500 cycles), the percentages of improvement in threshold sensitivity were 56%, 55%, and 55% respectively. For the higher frequencies (2000, 4000, and 8000 cycles), the percentages were 54%, 46%, and 36% respectively. A χ^2 of 1.22 was not significant, indicating that the lower frequencies did not show greater percentage of improvement than the higher frequencies.

Hearing Loss vs. Improvement

An important question is whether or not a greater preoperative loss yields a greater amount of postsurgical improvement. A Pearson product moment correlation coefficient of 0.58 was found between the 308 preoperative thresholds and the 308 postoperative thresholds. This coefficient suggests

a tendency for greater postoperative gain when there is a greater preoperative loss and vice versa. The size of the coefficient is reasonably high since there is a physiological limit on the amount of improvement that can be obtained from a conductive loss. Findings like those reported by Békésy [1] and Wever and Bray [7] show that removing the ossicular chain accounts for a maximum loss on the order of 60 db., which restricts the range of improvement. Postoperative improvement is restricted further by the presence of cochlear or retrocochlear pathology.

When the frequencies were ranked according to mean decibel improvement, an interesting result was found. There was a tendency for gross improvement to be correlated with frequency. Improvement tended to increase as frequency decreased, with 125 showing the greatest improvement and 8000 cycles showing the least improvement. To investigate this further, the sample was divided according to source (Juer's 22 cases vs. Scheer's 22 cases). The mean thresholds at each frequency were ranked according to decibel improvement in each subsample. A significant ρ-coefficient of 0.96 was obtained between the two rankings. Curiously enough, 125, 250, and 500 cycles were ranked first, second, and third respectively, with 2000, 4000, and 8000 occupying the last three ranks. With the exception of one reversal in rank order between the subsamples, there was a perfect inverse relationship between mean improvement and increasing frequency. Conversely, going from 125 cycles through 8000 cycles, there was progressively less over-all postoperative improvement. This result will require validation from a larger sample.

Age and Improvement

Is chronological age correlated with the degree of postsurgical improvement? The mean improvement over the seven frequencies for each patient was correlated with his chronological age by means of the rank difference correlation. That is, the 44 patients were ranked according to age and postoperative improvement. A nonsignificant ρ-coefficient of 0.08 was found. The ages ranged from 18 to 65, with a median of 38 years. Scheer [5] states that there is no correlation between age and degree of stapes fixation. This would lend support to our finding that age and degree of improvement from stapes mobilization were not correlated for preselected surgical cases. The presbycusic group would have to be considered separately.

Comment

The results are concerned with a statistical analysis of the effects of stapes mobilization on auditory sensitivity as measured by air-conduction, pure-tone thresholds. The sample under study was characterized by significant postoperative improvement. The mean preoperative thresholds differed significantly from each other, as did the postoperative thresholds. The median age was 38 years. The mean sample improvement was 22 db.

The Figure shows a parallel type difference between the pre- and post-thresholds. This represents the major effect of stapes mobilization on the transmission characteristics of the middle ear. A secondary effect was reflected by significantly greater gross improvement at 125, 250, and 500 cycles than at 2000, 4000, and 8000 cycles. This result is termed as secondary, since the differences were small compared with the over-all differences between preoperative and postoperative thresholds. This should give lower speech reception thresholds and is therefore important. The nature of the secondary effect was further evident when the postoperative frequencies were ranked according to mean improvement. There was an inverse trend between frequency and improvement. However, the effect was small and progressive and needs further study since the percentage of improvement was not significant when tested statistically.

The correlation of 0.58 between the preoperative and postoperative thresholds re-

vealed a tendency for greater preoperative loss to show greater postoperative gain. This should hold for cases equated for inner ear pathology.

A nonsignificant correlation was found between chronological age and degree of postoperative improvement. This finding did not cover surgical failures or consider the longevity of postsurgical improvement. The presbycusic age group should be treated separately.

One can hypothesize that a purely conductive loss should show a flat type air-conduction loss and a resultant parallel, postsurgical recovery as a function of frequency. This would imply that restriction of ossicular movement affects each frequency equally in the purely conductive loss. In the case of a mixed loss (conductive and perceptive), stapes mobilization should give a parallel improvement from 125 to 8000 cycles only after the perceptive component has been subtracted from the loss at each frequency either before or after surgery. Furthermore, a purely perceptive loss should not show any postoperative improvement after the Carhart correction factor has been applied. We are assuming that the correction applies to stapes mobilization cases as well as to fenestration cases.

Dr. Arthur L. Juers has commented on this paper (personal communication) and stated that our sample does not include cases of traumatic labyrinthitis, which occurs in about 0.2%-0.5% of stapes mobilization cases. These patients may show some decrease in high-tone perception, with the possibility of complete loss of cochlear function.

Summary and Conclusions

This study was concerned with a statistical analysis of the effects of stapes mobilization on the transmission characteristics of the middle ear. Forty-four improved patients were selected from two sources. The raw data consisted of pure-tone, air-conduction thresholds measured preoperatively and postoperatively at the octave frequencies of 125-8000 cycles. Despite sample size and probable differences in middle and inner ear pathology, noteworthy results were obtained.

The following are the major findings:

1. The major effect of stapes mobilization was a parallel type improvement as a function of frequency.

2. A secondary effect of surgery was a small significant trend toward greater gross postoperative improvement in the lower frequencies than in the higher frequencies. The percentage of improvement was not statistically significant. Improvement tended also to decrease as frequency increased in this sample.

3. Thresholds showing greater preoperative losses tended to improve more than those showing smaller losses.

4. There was a nonsignificant correlation between the age of the patient and his degree of postoperative improvement.

5. Since a purely conductive loss is generally characterized by a flat air-conduction loss, there is reason to believe that restriction of the stapes affects all frequencies equally, and thus mobilization of the stapes should result in parallel recovery at each frequency for these cases.

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Case Reports

Cerebrospinal Fluid Rhinorrhea in an Accident-Prone Individual

WILLIAM W. MONTGOMERY, M.D., Boston

The patient, an expert electrician, was born May 3, 1903, in Massachusetts. At the age of 15 he encountered his first serious accident while experimenting with gunpowder. Tinnitus and a perceptive hearing loss are the persistent symptoms resulting from the inevitable explosion.

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Massachusetts Eye and Ear Infirmary.

Before delving into the present illness, at least light note should be made of another incident in the patient's past history of accidents. Two days before Christmas, 1942, the patient was walking alone along a

Fig. 1.—Patient, age 53. Arrow points to the puncture wound.

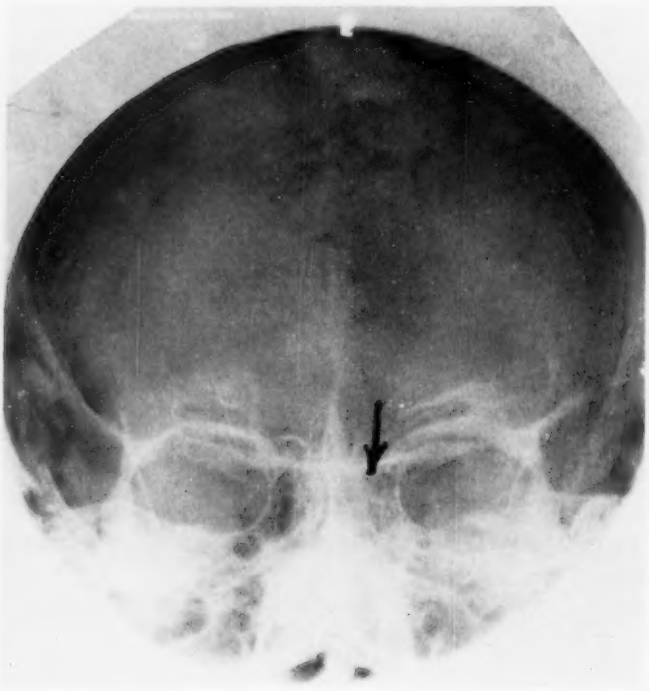

Fig. 2.—March 28, 1957. Left ethmoid dense.

roadway during a snow storm. He slipped on the ice just as a snow plow approached and became incorporated in a pile of snow for the next 150 ft. It was learned later that the driver mistook him for a Christmas tree. There was no loss of consciousness. Seventeen days of hospitalization were required for the treatment of a perforated tympanic membrane (healed spontaneously), scalp lacerations, and multiple ecchymosis. No x-rays were taken. A severe pain in the left cervical region which radiated down the left arm, and the inability to extend his head persisted after discharge from the hospital. Attempts to resume work were unsuccessful. In February, 1943, two months after the accident, he consulted an osteopath, who fortunately used only gentle massage to the back of his neck rather than gross manipulations. On May 1, more than four months after the accident, x-rays were taken. These showed a very obvious dislocation of the fourth and fifth vertebrae. A rather lengthy hospital stay was required for therapy consisting of traction wires to the head, then Crutchfield tongs, and then finally open reduction fusing C—3, C—4, and C—5. He returned to work in October, 1943, symptom-free.

On March 28, 1957, the patient, now age 53, was admitted to the Massachusetts Eye and Ear Infirmary after another unusual accident. He was working as a lineman. The line wires were held temporarily in a hook at the end of an 8 ft. length of spring wire ($\frac{1}{8}$ in.$\times\frac{1}{32}$ in.). You are probably anticipating an electrical shock or burn. No such thing! The spring wire broke and snapped back at the patient, striking him in the left eyelid. The pain was very severe, but only instantaneous. He cannot remember whether he pulled the wire out or whether it snapped free. After he was momentarily near unconsciousness, there remained only a small puncture wound in the left upper eyelid and moderate left epistaxis. Examination at the

Fig. 3.—April 2, 1957. Sinuses clear.

time of admission revealed a calm and rather pale patient, holding a blood-saturated handkerchief to his left nostril. He wore a hearing aid. Physical findings of note were as follows: a small puncture wound in the left upper lid (Fig. 1), which was dry; a scarred left tympanic membrane; a linear scar on the back of his neck, and left epistaxis. It was quite obvious at first glance that this epistaxis was blood-tinged spinal fluid. X-rays of the sinuses showed "left ethmoid dense" (Fig. 2). Ophthalmologic and neurosurgical consultations produced no alarming findings. The latter advised against a lumbar puncture. We all agreed on conservative therapy.

The patient was placed on absolute bed rest, large doses of penicillin and streptomycin, was skin-tested for and given 1500 units of tetanus antitoxin, and was very emphatically told that any nose blowing would lead to disaster.

The following morning the discharge of spinal fluid from the left nostril was crystal clear. A quantity of this fluid was collected by having the patient sit up in bed and lean forward. The only deviation from normal spinal fluid was the presence of a few red blood corpuscles. A temperature elevation of one degree persisted for two days after the accident. The spinal fluid leak ceased after three days. X-rays of the sinuses were clear on the fourth hospital day (Fig. 3). The patient was discharged from the hospital one week after the accident.

Summary and Conclusions

An account of the latest addition to a series of accidents indicates a definitely accident-prone individual. If studied, the personal aspects of this case might prove more interesting than the resulting lesions, at least from an industrial medicine viewpoint.

The perforation of the dura was probably in the region of the roof of the ethmoid, rather than the cribiform plate, because of the short duration of leakage.

Conservative therapy was sufficient for a full recovery.

It is of paramount importance that a patient with this type of injury be repeatedly warned against nose blowing, until healing has taken place.

243 Charles St. (14).

Granular-Cell Myoblastoma of the Larynx

CLARENCE D. HINTON, M.D., and MORRIS A. WEINBERGER, M.D., Washington, D. C.

The following case report is that of a laryngeal tumor of the type first described in 1926 by Abrikossoff.[1] This tumor, presumably composed of immature skeletal muscle elements, has been described variously as myoblastic myoma, granular-cell myoblastoma, myoblastoma, rhabdomyoma, and rhabdyome granulocellulaire. Although such tumors usually occur in sites where striated muscle is found, many have been reported from sites devoid of skeletal muscle. For this reason the exact origin of granular-cell myoblastoma remains obscure.[2] We are concerned here with a granular-cell myoblastoma of the larynx.

In October, 1954, MacNaughtan and Fraser reported the 14th case of granular-cell myoblastoma of the larynx and comprehensively reviewed the preexisting literature.[3] In view of the paucity of reported cases of granular-cell myoblastoma of the larynx, we feel that the present case should be reported. This case was diagnosed and treated at Freedmen's Hospital, Washington, D. C., in 1956.

Submitted for publication Feb. 3, 1958.

From the Division of Otorhinolaryngology and Department of Pathology, Howard University College of Medicine and Freedmen's Hospital. Clinical Assistant Professor of Otorhinolaryngology, Division of Otorhinolaryngology (Dr. Hinton); Assistant Professor of Pathology, Department of Pathology (Dr. Weinberger).

Report of Case

A 31-year-old Negro woman was admitted to Freedmen's Hospital, Washington, D. C., on April 17, 1956, because of hoarseness and dysphagia of approximately three months' duration. She had been seen previously at the Ear, Nose and Throat outpatient clinic where she was found to have a small nodular mass superior to the left false vocal cord and just anterior to the left arytenoid cartilage. She was admitted to the hospital primarily for the removal of this lesion. During this hospitalization additional positive findings were hyperemia of the vocal cords and a chronic tonsillitis. All laboratory studies, including urinalysis, hematology, serology, and blood chemistry were within normal limits.

Her past history revealed a previous hospitalization at Freedmen's Hospital in 1945, at which time her serology was positive, and she was treated for syphilis.

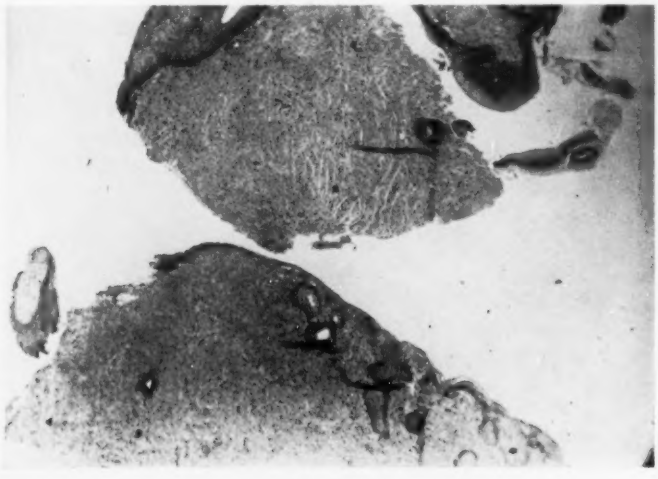

Fig. 1. — Low-power photomicrograph showing two pieces of laryngeal neoplasm. The surfaces are partly lined with stratified squamous epithelium, beneath which masses of adjoining granular cells are seen. (Reduced about 29% from mag. × 50.)

An attempt to remove the laryngeal lesion on the fourth hospital day was unsuccessful because of the patient's nervous and uncooperative state. She was discharged from the hospital two days later and readmitted for surgery on June 28, 1956. On the following day the lesion was removed under topical anesthesia (1% tetracaine [Pontocaine]). The laryngeal tumor was quite firm and felt somewhat cartilaginous to the bite of the laryngeal biopsy forceps. It was spherical in shape, measuring about 0.5 cm. in diameter.

The pathologist's report was as follows: "The gross specimen consisted of two roughly spherical portions of firm, grey tissue which measured in aggregate 0.5×0.3×0.2 cm. Microscopically, the fragments were partially lined with stratified squamous epithelium. One fragment had a lining consisting partly of respiratory type epithelium. The stratified squamous epithelium focally was thinned and had a keratotic layer (leukoplakia). Beneath the lining mucosa were a few solid nests of squamous epithelial cells (Figs. 1 and 2), one of which resembled an epithelial pearl. Also present submucosally were laryngeal gland ducts seen in cross section. Most of the submucosa was occupied by large, round, oval, or elongated cells which had abundant granular eosinophilic cytoplasm (Fig. 3). The nuclei of these cells were quite variable in size, shape, and staining intensity. They were round, oval, or elongate, and some appeared moderately hyperchromatic. Most of the nuclei were eccentric (Fig. 4). There was a tendency for the granular cells to collect in bands or bundles. Blood vessels were not conspicuous in

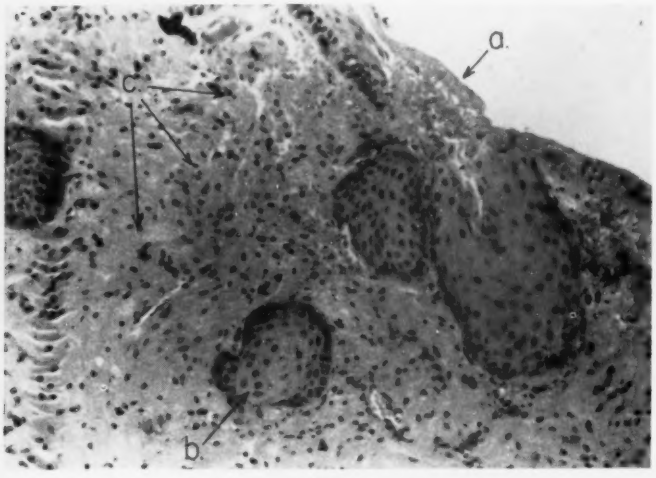

Fig. 2. — Low-power photomicrograph of portion of lower fragment in Figure 1, showing (*a*) keratotic layer on surface squamous epithelium; (*b*) island of squamous epithelial cells penetrating deeply into the neoplasm (pseudoepitheliomatous hyperplasia); (*c*) granular cells comprising the bulk of the lesion. (Reduced about 29% from mag. ×200.)

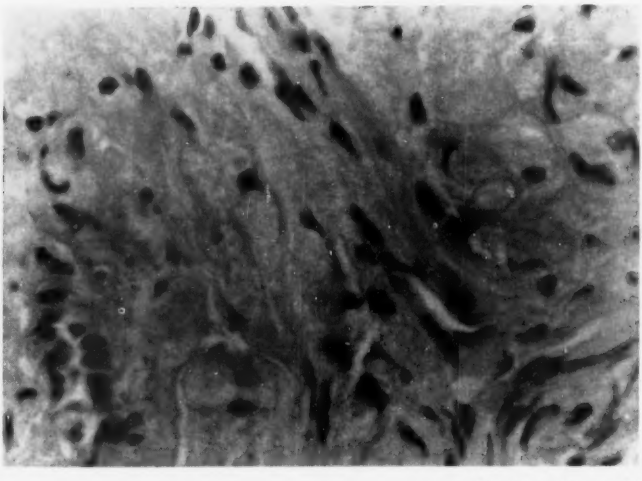

Fig. 3. — High-power photomicrograph of neoplasm showing granular cells with pleomorphic eccentric nuclei and abundant granular cytoplasm. (Reduced about 29% from mag. ×700.)

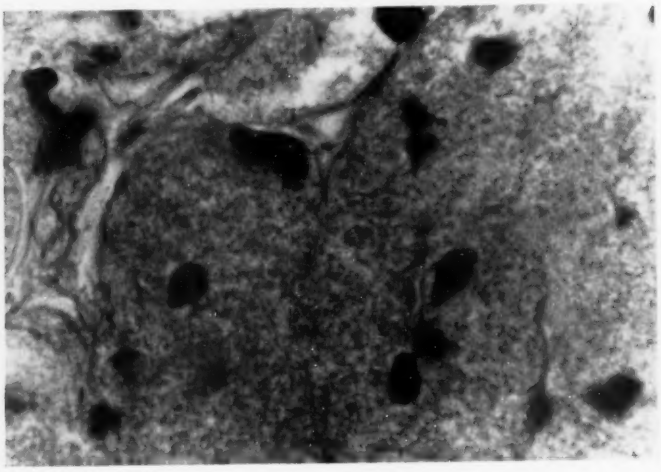

Fig. 4.—Oil-immersion view of several granular cells showing minute cellular structure. The granular character of cytoplasm is clearly depicted. (Reduced about 20% from mag. × 1530.)

the lesion. Skeletal muscle fibers and granular cells were noted." The pathologist's impression was "granular cell myoblastoma of the larynx."

Direct laryngoscopy at the time of this operation revealed two small nodules on the opposing surfaces of the free edges of both true vocal cords, at the junction of the anterior and middle thirds. These were very small and had the appearance and location of benign vocal nodules. We therefore elected to treat them conservatively for a period of time before considering excision.

On July 3, 1956, a tonsillectomy was performed under local anesthesia (1% procaine [Novocaine]). Secondary post-tonsillectomy hemorrhage occurred, which required the return of the patient to the operating room, where hemostasis was effected. Her convalescence thereafter was uneventful. She was discharged on July 4, 1956.

On Sept. 11, 1956, the patient was again admitted to the hospital, this time for the removal of the vocal nodules which had failed to respond to vocal rest and local treatments. Under topical anesthesia, the patient was again so uncooperative as to make removal of the small nodules an impossibility.

Approximately 15 months elapsed before the patient consented to removal of the vocal cord lesions. They were excised on Jan. 24, 1958, and proved to be inflammatory polyps. There was no evidence of the previously excised granular-cell myoblastoma.

Comment

This patient's main symptom of hoarseness was due to small "vocal nodules" which were present on the free edges of both vocal cords. The resected laryngeal lesion in this case was asymptomatic and was discovered on indirect laryngoscopy for hoarseness. In contrast to most of the 14 cases reviewed by MacNaughton and Fraser, this lesion was not located on a vocal cord, but, rather, above the left false cord, just anterior to the left arytenoid cartilage. The relative youth of the patient (31 years) corresponds to the majority of previously reported cases. Of 13 such cases, having age data, 9 were 40 years of age or younger. The sex incidence in 13 reported cases with data showed 9 males and 4 females. No final conclusions are permissible regarding age and sex incidence in view of the small number of reported cases of granular-cell myoblastoma of the larynx. The present lesion was a pea-shaped, broad-based nodule, 0.5 cm. in diameter and similar in description to seven of the previously reported lesions. It was easily accessible for removal with laryngeal forceps. The patient was free of recurrence 19 months after surgery. If completely removed, the laryngeal granular-cell myoblastoma seems to have little predilection for recurrence, regardless of the mode of removal. Only two of the reported cases developed recurrences.[3]

Pathologically, the stratified squamous epithelium overlying the lesion showed a characteristic pseudoepitheliomatous hyper-

plasia, mistaken by the early workers for cancer.[4,5] The granular cells composing the lesion showed no features of malignant neoplasia. The location of the tumor subjacent to laryngeal mucosa (a site normally free of skeletal muscle) and the absence of any normal skeletal muscle fibers in the histologic preparation raise some doubt about the origin of this lesion in skeletal muscle. It was not possible for us to trace the pathogenesis of this lesion from the histologic section.

1116 W St. N. W. (9)

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Clinical Notes, New Instruments and Techniques

Room for Maneuver in Peroral Laryngoscopy

Presentation of a New Multipurpose Laryngoscope

ROBERT B. LEWY, M.D., Chicago

The physical restrictions imposed by the walls of the laryngoscope tube reduce visibility and freedom of access to many laryngeal lesions. The diameter of the mouth, unlike that of other body orifices, is large enough to permit solutions to this problem. Some such were developed years ago with only a modicum of success, which probably explains their virtual disappearance from the surgical scene. Prior to 1916, Bruening, Mosher, and Ingals[1] all developed laryngoscopes with one side open to give more room for instrumentation. These were hand-supported, thus leaving one hand of the operator for surgical purposes. In addition, the surgeon's unbalanced crouch imposed a handicap.

The suspension apparatus was pioneered by Killian and others, and finally securely established by Lynch.[2] This created an open field which made extensive peroral surgery a practical reality. As Lynch's followers have proved,[3] the use of this apparatus permitted accurate two-instrument dissection, fairly extensive use of cautery, and the removal of large tumors. Reported were a series of malignant tumors successfully removed and destroyed with the modality popularized by Lynch. In addition, accurate two-instrument dissection was applied to vocal cord edges. Benign tumors were removed by dissection rather than avulsion.

Heinberg[4] recently revived interest in the open side laryngoscope, presented in tandem with a chest-supported laryngoscope holder,[5] enabling a bimanual freedom for essential tasks. The method permits readily used and easily transported instruments.

I am herewith presenting a multipurpose laryngoscope for similar and additional purposes. This particular instrument is longer and has a flare tip which enables the operator to reach and expose the anterior commissure. It is of larger diameter and gives greater room for the combined use of two instruments. An arytenoidectomy was performed without difficulty using this instrument.

The laryngoscope is designed for the following applications:

1. Standard manual laryngoscopy.

Submitted for publication June 17, 1958.

From the Department of Otolaryngology, University of Illinois College of Medicine, and Michael Reese Hospital.

Fig. 1.—Multipurpose laryngoscope designed by the author.

Fig. 2.—The multipurpose laryngoscope with the new model gear, leverage laryngoscope holder with the side drive.

2. Use with the laryngoscope chest support for neoplasm removal; biopsy; arytenoidectomy; two-instrument dissection.
3. Aiding the introduction and passage of a bronchoscope, breathing tube, or anesthesia tube.
4. Anterior commissure work.
5. Use with topical anesthesia. Its use with general anesthesia and relaxants is practical but rarely necessary.

Assistance was given by Dr. S. J. Pearlman in clinical evaluation.

Instrument manufactured by V. Mueller & Company.

25 E. Washington St.

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News and Comment

ANNOUNCEMENTS

American Association for Cleft Palate Rehabilitation.—The 17th Annual Convention of the American Association for Cleft Palate Rehabilitation will be held in Philadelphia, at the Sheraton Hotel on Thursday, Friday, and Saturday, April 30, May 1 and 2, 1959.

This Association is composed of medical, dental, and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates.

New Speech Journal.—After 38 years of continuous publication, *Talk* Magazine has been discontinued. It will be replaced by another Journal, *Logos*: The Bulletin of the National Hospital for Speech Disorders. The new periodical will be devoted exclusively to the publication of scientific papers in the entire field of human communication. Experimental, theoretical, and clinical papers on language, speech, voice, hearing, acoustics, psychology, psychiatry, neurology, and semantics, with special reference to physiology, pathology, and therapy, will be contributed by specialists in these fields.

It is intended that no restrictive editorial policy should impose any limitations on the subject matter to be presented. Since the Bulletin is from America's largest diagnostic and treatment center for speech and voice pathology, there will be a certain emphasis in *Logos* on experiences and findings made by the staff members of this specialized institution. However, the pages of *Logos* will be opened to all contributors who may wish to exchange their opinions with this institute.

The first issue of *Logos* is expected in October, 1958. Subsequent issues are planned on a semiannual basis—one each spring and autumn. All communications regarding submission of papers, reprints, subscriptions, or other inquiries should be addressed to the Editor, Logos, National Hospital for Speech Disorders, 61 Irving Place, New York 3.

Progress in Otolaryngology

Functional Examination of Hearing

Summaries of the Bibliographic Material Available

ALFRED LEWY, M.D.; NORMAN LESHIN, M.D.; SHERMAN L. SHAPIRO, M.D., and LEO A. SATZ, M.D., Chicago

Introduction

The material herein represents excerpts from the literature from October, 1956, to October, 1957. This year the German literature was abstracted by Dr. Satz. An attempt was to cull from the ultrascientific reports of the acoustic physicists such material as was thought useful to the practicing otologist, who may not have the extensive facilities required for many of these investigations.

Hearing Aids

Ewertsen and Ipsen [1] state that measurements show that the reproduction of high frequencies is better when an ear mold with a short and wide canal is used rather than one with a long and narrow canal. With the so-called secret ear the high frequencies are cut off more markedly.

Naunton [2] examined a group of partially deafened adults after varying periods of hearing aid use to determine whether the hearing aids had led to a deterioration of unaided hearing. No significant change in hearing could be demonstrated.

Pattee and Cary [3] discuss the use and abuse of hearing aids. By using proper speech testing in addition to pure-tone threshold testing they have been able to disclose improperly fitted hearing aids in many persons. With complete speech and pure-tone audiometry tests 65% of 79 children tested were found to have improperly fitted aids and were advised to make some change. The advice given ranged from changes in the method of wearing the aid to a purchase of a new hearing aid. They stress the importance and the responsibility of the otolaryngologist to properly equip himself to give good and adequate advice about the many problems involved in the use and selection of a hearing aid. They previously described an economical method of speech testing which one can carry out in his office (Free-Field Audiometric Speech Testing in a One-Room Testing Arrangement, A. M. A. Arch. Otolaryng. 64:63 [July] 1956).

A prospective hearing aid user is more likely to adjust well and quickly if he is given information on the bad as well as the good features of the aid as applied to his specific case. Holcomb [4] feels that the audiologist can render real assistance as an adviser and counselor during the difficult breaking-in period. He can prepare the patient psychologically. The rapid, automatic, subconscious interpretation of the partially new language heard through a hearing aid can be achieved only by months of constant use under real conditions.

Clinical Tests

Jakobi [5] discusses the various auditory tests used in children of different ages to determine their hearing function.

Previously, he states, it was thought that hearing tests could successfully be performed only in children of school age. They were tested with the help of test words, tuning forks, or pure-tone audiometry.

Received for publication March 7, 1958.

In infants and small children only rough audiometry tests had sufficed. They were done merely to affirm or to exclude deafness or hardness of hearing. These rough tests commonly in use are the auropalpebral reflex of Bechterow or the cochleopupillary reflex of Holmgren. Another test is the vestibular function test, well applicable in small children. Complete deafness goes along with vestibular hypo- or nonfunction; while presence of normal vestibular function points more toward presence of residual hearing in children (hereditary and congenital deaf-mutism). However, in daily routine examinations in small children one tries first all kinds of noise-making contraptions (bells, trumpets, buzzers, etc.) to determine the presence of hearing in a child. This method, of course, is subject to many errors. Playing with musical toys was used in children for qualitative and quantitative determinations of hearing for many years. Arnold created an electric contrivance (bells, buzzer, horns, etc.) for testing of hearing in children. One person handles the noise makers; another watches the child's reactions. From the distance, pitch, and loudness of the respective musical toy one tries to deduce the quality and quantity of hearing function present in the child. Results are not always very accurate.

Only the introduction of modern audiometry has enabled us to obtain useful threshold of hearing in children, using special techniques and observing the child's reaction. Special impetus was given to improved and more exact auditory testing by hearing-improving and hearing-preserving operations and procedures as well as by the introduction of auditory rehabilitation by making use of the hearing rests in the hard of hearing and deaf-mute children. Precise threshold measurings of hearing by speech or pure tones were now needed to help one evaluate any gain attained by the above-mentioned procedures; also to determine the presence of hearing rests in hearing defective children. This may help the teacher to offer the child the first auditory impressions in the frequency he best hears. Thus systematic auditory training may be instituted. Amplification makes it possible for the child to hear his own speech and thus learn a normal language. Further auditory training may enable the child to go to a regular school or a special vocational school.

Since many hard of hearing children can be helped by auditory training, it becomes necessary to make useful qualitative and quantitative hearing tests so that early auditory training rehabilitation may be instituted (first to second year of life).

In children of 7 or 8 years of age auditory test as in the adult are quite possible to remove any defect in hearing.

In children below this age to uncover defective or residual hearing one has to resort to the evaluations of the child's reaction and associations to acoustic stimuli (electroacoustic hearing tests).

In 1942, Bloomer tried to test children's hearing with the help of musical toys which produced sounds in the audiometer. Guilford constructed a similar apparatus in the manner of a little house with people and animals in it which reappear with the help of the audiometer. The eight figures hidden in it represent the main tones of the audiometer.

Westlake tried to test children with speech audiometry after the mother had exercised with the child and prepared it for the test.

Marcus made EEG curves on sleeping children following acoustic stimulation. However, by none of these tests could threshold measurements be attained.

In 1947, Hallpike tried to test children with the help of a peep show. This test is applicable to children from the third year of life on.

Jakobi created his own contrivance after the model of the peep show, a children's movie consisting of fairy tale films and children's scenes. It rotates, driven by a special motor, and is connected to an audiometer. The child presses a button. After the flashing of a light and sounding of a test tone, the pictures appear. After practice the child finds that the awareness of

the test tone alone is sufficient to make the moving pictures appear. This method is called *film audiometry*. After the child has been conditioned, a threshold audiogram is tried. This method has proved effective in cases where other methods for threshold measurements failed. With this method psychogenic deafness in older children could be uncovered and thresholds made.

To test yet smaller children audiometrically or to attain a totally objective hearing test from adults and children the author used the *psychogalvanic skin resistance test*. This test was applied in children of 1 to 2 years of age, in deaf-mute adults, in psychogenic deafness, and in malingerers.

From all the methods of testing hearing in children the method of *Spiel audiometry by Barr* proved the best in Jakobi's clinic (rarely applicable in children under the age of 3 years). Occasionally he used speech audiometry in children of preschool age. He used the appropriate words according to the age of the child. Sometimes he used commands or questions with answers at different intensities. He has his own groups of words applied to children of different ages (2 to 6 years); four word rows, each containing 10 words, according to the mental development of the child.

He also has sentences used as commands or questions and answers through the amplifier at different intensities to find thresholds in speech audiometry in small and preschool children. He calls it sentence-speech audiometry. Six groups of PB sentences with five commands or questions each are applied.

In summary he concludes that

1. Through early testing in the very young children with possible hearing defects early therapy can be instituted (tubal, physical, radiological, conservative, and operative ear therapy). The auditory threshold curves may predict success or failure of the therapy.

2. The uncovered residual hearing with the help of amplifiers, hearing aids, etc. must be utilized to give the child as natural a speech as is humanly possible.

3. For the small child (up to age of 3) and the less intelligent older child, for the adult malingerer and for one with psychogenic deafness the use of the objective psychogalvanic skin resistance test is recommended.

4. The simplest and in most cases also the most successful tests for children, age 3 to 6 years, are the Spiel audiometry tests by Barr.

5. His film audiometry tests were very valuable in play-fatigued but technically minded children. Even psychogenic deafness in older children could be diagnosed.

6. For speech audiometric testing of preschool children and smaller ones, he has for each age, according to its intelligence, a group of words as well as a group of sentences created, and used with success.

According to Oeken,[6] the audiometric hearing test methods permit a much better evaluation of hearing in each individual ear than the previous tuning fork tests. A good sound protection is already attained by the rubber padding and the tight fitting of the head phones.

In greater difference of hearing between the two ears this protection is inadequate. Masking of the better hearing ear becomes necessary. Many types of noises have been devised to accomplish it. With the properly selected noise and intensity it is possible to exclude the better hearing ear from the testing and thus to measure the auditory performance of the worse ear. It works very well in air-conductive testing in all cases. In bone-conduction examinations difficulties arise. There are the rare cases where in spite of the best masking technique a threshold curve for the worse ear cannot be determined. This may occur in patients in whom in the better ear there exists a high-grade defective conductive hearing. In these cases, either one does not succeed in conveying the masking noise to the inner ear in sufficient intensity via the air-conductive apparatus—in which case one cannot measure the bone conduction of the worse ear since it is overheard in the better hearing ear—or the masking noise, if it is of very strong intensity, becomes converted into bone sound and then masks the tested ear, too. Such an auditory picture may be found in otosclerotics. Especially here errors in evaluating the bone conduction of the worse ear may lead to disastrous results, since it is the worse ear that is usually

operated on as long as its inner ear function appears adequate.

In the past few years speech-audiometric hearing test methods have been introduced and improved. One assumes, of course, that over-hearing does not play such a big role in speech audiometry with its complex tests and test words as in tone audiometry.

To attain a comparison of over-hearing in tone and speech audiometry the author made speech-audiometric investigations on 20 unilateral deaf patients. In the tone audiometry the shadow curves in unilateral deafness measured via air conduction on the deaf ear were about 50-60 db. lower than on the healthy ear. (Langenbeck and Jorgensen think that in "over-hearing" the air sound is converted into bone conduction and carried to the hearing ear in this way.)

In evaluating 12 speech-audiometric shadow curves of unilateral deafness with normal hearing on the hearing ear, Oeken found that the "over-hearing curves" measured from the deaf ear out were displaced an average of about 60 db. toward the higher intensities. Observation of the distance of the curves shows in average a parallel course to the curve of the hearing ear. The almost purely parallel displacement indicates that at the examination from the deaf ear out he found that the understanding of speech climb is practically just as fast to 100% as in the hearing ear. The comparison with the tone-audiometric shadow curves shows a close agreement between the curve distances. Further tests along the same lines revealed similar results, which suggests that the over-hearing occurs not via air conduction but via bone conduction.

In summary he concludes that the speech-audiometric investigations on unilaterally deaf people has revealed that the over-hearing curves, as in tone audiometry, lie about 60 db. below the curves of the hearing ear.

The shadow curves in these cases showed a remarkably parallel course.

The climb in speech understanding occurs, therefore, practically just as fast as in the hearing ear.

Tight packing of the auditory canal with cotton, etc., makes no change in the distance between the shadow curves, so one must assume that the "over-hearing" occurs via bone conduction.

The findings in unilaterally deaf patients with sound-conductive type of hard of hearing in the hearing ear confirm this assumption, since here the distance of the overhearing curves is much smaller.

One must conclude from these investigations that in speech audiometry, too, masking of the better ear is needed, and that, especially in unfavorable cases of asymmetric loss of hearing, speech audiometry, too, does not give sure information about the hearing of the worse ear.

Jatho[7] summarizes his scientific paper as follows:

The defective hearing in inner-ear impairment is especially marked by the presence of loudness balance. This defect in hearing causes in the hard of hearing a pronounced auditory distortion of the speech sounds.

It is attempted in this paper to determine graphically the form and degree of the auditory distortion in the impaired ear by plotting the energy spectra of the individual vowels for a given sound-perception hearing impairment with loudness balance. In this way one arrives at the virtual loudness spectra of the investigated vowels which always deviate markedly from the original energy spectrum according to the type of the defective hearing and of the loudness balance.

Recruitment is interpreted, in contrast to the up-to-date concept of a pathologically increased hearing acuity, as an expression of a pathologically coarsened acoustic resolving power which finds its parallel in steep black-white gradation of light-sensitive photographic layers.

The importance of the sonority equilibrium test (recruitment) for the differential diagnosis of deafness through cochlear, pre- and retrocochlear lesions was described by Segal and Hociota.[8] There were 45

patients with precochlear lesions (such as cerumen), 12 meso- and epitympanitis, 10 tubal catarrh, 8 radical mastoid operations, 6 otosclerosis. The recruitment test was negative in 97.8% of these cases. There were 17 with Ménière's syndrome, 3 acoustic trauma, 2 labyrinthitis. The test was positive in 90% of these cases. There were eight patients with acoustic neuromata, one meningioma, two acoustic neuritis, six parietal cerebellar arachnoiditis. In this group the test was negative in 88.2%. The test could not be carried out in symmetrical bilateral partial deafness or in total deafness.

Meyerson[9] developed a verbal audiometric test for measurement of auditory acuity in infants and children. The recordings consisted of two parallel high-fidelity forms and one form with emphasis on the higher frequencies, suitable for individual or group testing of primary grade or preschool children. Also three forms were recorded for children in Grades 4 to 12.

The reproducing equipment consisted of a high-fidelity reproducer and amplifier, matching attenuater, voltmeter, and 10 pairs of earphones. Spondaic words were used, selected for speech sound distribution and familiarity. Each stimulus word was preceded by a three-word carrier phrase. In the forms for younger children the form directed them to place a number 1 to 12 beneath a specified picture. Intensity was attenuated 3 db. from the previous stimulus. Those who failed on the 9th or 10th stimulus fell one standard deviation or more below the mean for their group in hearing for speech. The reliability of the method was determined by test-retest and alternate form administration.

On 432 preschool and 286 Sixth Grade ears it was found that there were no significant differences between the experimental and reliability studies. Children in an intellectually slow class had poorer hearing than regular classroom children.

Söhoel[10] discusses the course of bone conduction after the fenestration operation. Apparatus and procedure are described. Pressure as well as site of application affect bone conduction. The author prefers frontal application. Thirty-five otosclerotic patients were examined before and after operation. All had been examined preoperatively and daily for two weeks thereafter: some three, four, and five weeks postoperatively. Eighty per cent reported improvement in bone conduction the first day after operation, and they had eventually a 24 db. gain in the speech area. It is presumed that such primary gain indicates a good prognosis.

Anderson, Holmgren, and Holst[11] suggest an objective method of testing middle-ear function. Vibrations of 200 to 2000 cps are applied to the skull. A sound pressure develops in the auditory canal and is recorded as a frequency function. The authors believe that the sound pressures are affected by the dynamic properties of the tympanic membrane, which are assumed to be related to the status of the middle ear. The theory is supported by investigations of normal and pathologic ears and a model skull.

Hearing loss in aphasia is discussed by Stansell.[12] Ninety aphasic patients in the Minneapolis Veterans' Hospital were examined. Eighty-eight per cent had hearing losses, which averaged greater in the older group and also averaged greater in the cerebrovascular or tumor group than in the traumatic group.

Auditory tests on newborn infants were made by Wedenberg.[13] Twenty children up to 10 days old, and one Rhesus monkey, aged 3 days, were tested by the auropalpebral reflex; intensity of sound needed to awaken the child from sleep. A pure-tone audiometer with a loud speaker was used. In normal cases it was possible to obtain the auropalpebral reflex at 110 to 115 db. If they slept too soundly, they could not be aroused by the tactile palpebral reflex either. The author expects to apply control audiometry on the same children at 3 years of age.

Twenty-two patients with unilateral Ménière's disease with abrupt loss of acuity

for high frequencies were examined for recruitment by Hedgecock.[14] Recruitment was present in all the diseased ears for all tones, and for 4000 cps in the well ears. Speech reception was seriously impaired in the diseased ears and only slightly in the better ears.

Moulden [15] discusses the Weber's test and recruitment. He prefers c-2, 512 d. v., placed on the vertex; after checking the ear to which the sound is referred, recruitment is indicated if the sound is heard first in one ear, then in the middle, and then to the other side during the same application. The test is applicable if hearing is unilateral or markedly different in both ears.

Ballantyne [16] conducted an investigation of the Denes-Naughton difference limen technique. Twenty-two subjects were tested, and it was found that the presence of recruitment was found in normals; on repetition the tests were not necessarily consistent. It was concluded that this technique is not reliable.

In pure receptive lesions a relation was proved between hearing loss by air and recruitment degree. Miskolczy-Fodor [17] claims his method is accurate even in cases of mixed deafness. The article does not lend itself to abstracting but should be read in the original, which is published in English.

Lauritz Lund-Iversen [18] investigated noise and hearing conditions on board Norwegian motor torpedo boats and submarines. In the engine room of motor torpedo boats sound pressure was 102 to 124 db.; in submarines (Diesel engines) sound pressure was 110 to 112 db. Efficient hearing protection and other methods were emphasized, and routine entrance and interval checking advised.

Rubenstein and Klein [19] attempted to evaluate the Weber test. Where both ears have loss of hearing, it may be difficult to determine for purposes of fenestration which ear should be selected for surgery in cases of mixed deafness. The authors examined a considerable number of cases, excluding those whose audiometric findings were inconsistent or otherwise unreliable on repeated tests.

Wiegers [20] states that Holmgren as early as 1939 pointed to the possibility of intraoperative audiometry. At the present time it has become a common procedure in stapes mobilization operations.

To soundproof the operating room and to install special testing equipment would be very costly, they feel; besides the room would always be an acoustic source of errors. Testing with whisper and conversational voice or with tuning forks is inadequate. They therefore used for intraoperative audiometry a simple audiometer with a tightly fitting air-conduction receiver and a light signal button. The transmitting of sound from the sound-producing receiver to the bone is prevented by a tightly fitting cylindrical sponge rubber padding on the receiver. The well-insulated air-conduction receiver is then wrapped in a sterile cloth for use for the operator, as needed during the operation. It is put tightly over the ear or even over a retroauricular wound; thus an exact threshold test over all frequencies is possible during the operation. He mentions that Goodhill carried out similar investigations during Rosen operations.

On their surgical cases they usually make preoperative, intraoperative, and postoperative auditory tests. The preoperative tests consist of pure-tone threshold audiometry, speech audiometry, and examinations with sound probe to determine first the type and localization of the auditory lesion. During the operation the intraoperative testing is performed as needed. The well-padded air-conduction receiver insulates the tested ear adequately in presence of moderate external noises. Shadow curves start first between 60 and 70 db., so no marking is absolutely needed.

The frequencies tested are 1000 cps, 4000 cps, and 250 cps; in complicated cases all frequencies are tested. Patient answers with the signal light when threshold of frequency is reached. The position of the intraoperative hearing curves depends very

much on the centrally depressing effect of the preoperative medication, and the individual constitution of the patient.

Auditory thresholds of normal hearing and hard of hearing patients were compared *after* the same preoperative medication and *before* the preoperative medication. It has been found that in some drugs the auditory thresholds are unchanged; in other drugs there is a reduction of 10-20 db. in the auditory thresholds.

They draw the conclusion that intraoperative audiograms can not, therefore, be compared with audiograms taken before or after the premedication.

Naunton [21] investigated the occlusion effect resulting from the use of a pair of standard-type air-conduction receivers in association with a frontally applied bone-conductor receiver, and the relation between air-conduction and frontal bone-conduction hearing losses on a group of 285 deaf and normally hearing subjects. Those with normal middle ears showed average occlusion effects of 14.2 db. at 250 cps, 13.6 db. at 500 cps, and 3.3 db. at 1000 cps. The subjects with abnormal middle ears showed negligible occlusion effects at these frequencies. Occlusion effects were negligible for all ears at 2000 and 4000 cps.

Pathological middle ears may, for convenience, be regarded as being in an occluded state. If tests are made with occluded ears, using an audiometer calibrated on occluded normals, a more valid picture of the nerve part of the mixed deafness will result. To avoid correction factors bone-conduction audiometers should be recalibrated to give measurements relative to the occluded normal and by making bone-conduction measurements in the occluded state. Additional unoccluded tests should be made at 250 and 500 cps. The comparison of the two types of test serves to confirm or deny the presence of a middle-ear lesion. If a frontal bone-conduction receiver is to be used with a standard audiometer whose bone-conduction test circuit is calibrated for mastoid tests, the instrument must be recalibrated on a group of normally hearing persons. An occlusion effect demonstrable within the limits of the writer's technique will indicate with a high degree of accuracy the presence of a normal middle ear. Naunton feels that much is gained in clinical bone-conduction measurements by occluding the ears and by using a bone-conduction receiver applied to the frontal rather than to the mastoid.

Schimizer and Nakamura [22] describe a variation of the audiometer peep show test for testing preschool-age children, using lantern slides. A conditioned reflex response is established by showing a picture on the screen when a tone is heard and a button is pressed. The picture will not appear when the button is pressed without the sound being heard. Different pictures are used so as to keep the interest of the child. Since the child may become tired, it is suggested that the hearing threshold be rapidly established first for 250, 1000, 4000, and 8000 cps and then extended to all the frequencies, if the child will continue to cooperate, or at a subsequent test. The equipment is simple and the test is easily applied. The writers suggest that the tester have an interest in children and be skillful in handling them.

Palva [23] gives a short review of papers dealing with self-recording audiometry and recruitment. He tested 17 normal subjects and 56 perceptively deaf persons. The average normal threshold amplitude varied between 7 and 10 db. Smaller than 5 db. excursions are occasionally found in normal hearing persons and the upper limit seldom exceeds 20 db. The importance of the determination of the loudness function with direct recruitment function is stressed. Asymptotic recruitment is, as a rule, associated with small excursions. Many cases with straight-line recruitment function showed larger than 5 db. amplitudes, and patients with delayed recruitment showed excursions comparable with those of non-recruiting subjects. In the perceptively deaf cases with no recruitment there were some values on the dividing line while the

majority were larger than 5 db. At frequencies at or above 2000 cps there is a good correlation between small threshold amplitude and recruitment. At the low frequencies one cannot predict the loudness function on the basis of threshold tracings. These findings indicate that direct loudness recruitment tests cannot be supplanted by the self-recording technique.

Palva[24] reviews various indirect recruitment tests, such as tests for intensity and frequency difference limen, masking, auditory fatigue and adaptation, self-recording threshold audiometry, and speech intelligibility. He concludes that wherever it is technically possible recruitment should be measured with direct tests. In cases with nearly the same threshold loss in both ears and at all frequencies self-recording threshold audiometry and speech discriminative tests were found most reliable for predicting the presence of recruitment.

Carhart[25] presents a test for auditory adaptation at threshold which was developed in 1954 at Northwestern University. This is known as the Threshold Tone Decay Test. The purpose of the test is to determine the phenomenon of "relapse." A pure tone is presented at threshold for a minute unless it fades before that time. In that event the intensity is increased 5 db. No rest period is allowed. The procedure is repeated until the tone is heard at one level for a full 60 seconds or until abnormal adaptation at threshold has been demonstrated. This test, as well as another practical method for isolating auditory relapse, holds the key to a new precision in the differential diagnosis of sensorineural lesions of the hearing mechanism. Clinical cases show varied configuration of response to this test. A systematic study of the phenomenon can probably lead to classification of sensorineural involvements into new clinical subgroups.

Tests on the identification of sound level and matching from sample were made by Pollock.[26] A number of methods were used to determine which gave the most accurate results in identifying the sound level. The method in which the subject was permitted to hear the sound being tested, and allowed to switch back and forth with other intensities for comparison, gave the most accurate results.

Hood[27] discusses the difference between fatigue and adaptation (and pathological adaptation) of hearing. Auditory fatigue is threshold elevation for test tones following sustained stimulation by these tones of high intensity. Measurement of adaptation can be done by binaural loudness balances of loud tones of identical frequency. After adaptation of one ear to a sustained loud tone the subjective loudness level will show a decrease of about 30 db. In pathological adaptation the effect of brief intermittent stimulation may be normal, while sustained stimulation results in pathologic relapse of the response to a subnormal adapted level. Ménière's disease produces abnormal adaptation of the affected ear. The finding supplements the results of testing for recruitment, the two inseparable signs of end-organ disease. Abnormal adaptation calls for a special design in audiometric tests.

Jeffress, Blodgett et al.[28] report on 30 experiments in masking which they interpret. The experiments were checked on two electronic device models of the human ear that served as virtual extensions of the ear, which could be experimentally adjusted and their behavior studied.

Portmann and Portmann[29] contribute another article on the use of the psychogalvanic reflex in otology. They state that by means of this technique it is possible to obtain results within 2-5 db. of threshold in adults and older children; within 10 db. in children 4 to 7 years of age; within 15-20 db. in children 2½ to 4 years of age, and within 20-30 db. in infants under 2½ years old. They list three indications for use of this method: in psychogenic deafness; in simulators, and in children too young for "peep-show" and other similar methods of audiometry. They warn against

two pitfalls: First, rigorous attention to details in the technique of performing the test, as otherwise the results can be misleading, and, second, against taking the results too literally in children, where only an approximate hearing curve is possible.

Beretta[30] discusses the audiometric aspects of Ménière's disease. He considers that the vestibular symptoms, particularly in the intervals between attacks, do not give sufficient information for the diagnosis of labyrinthine hydrops, which is the modern concept for this condition. He also calls attention to "cochlear deafness" of Wright without vertigo, which also represents a malady arising from a dilatation of the endolymphatic system. The fundamental diagnosis of labyrinthine hydrops rests chiefly upon the audiometric picture of a flat or lightly rising curve up to 5000 or 6000 cycles, reduced bone conduction on the involved side, recruitment by the Fowler method, or lowered discrimination threshold with the Luscher method. To aid in the diagnosis the author has used the injection of polyvinylpyrrolidone both during and between attacks. This is a substance of high molecular weight in the form of a white powder which is used in 3.5% solution to combat shock and circulatory collapse. The technique for diagnosis of labyrinthine hydrops is to inject 50 cc. of 10%-15% solution in normal saline. In acute attacks this terminates the vestibular symptoms immediately. Whether it is performed during an attack or between attacks, there is noted in 24 hours a rise in the threshold which can exceed 30 db. as well as a marked improvement in bone conduction which lasts for a few days; the effect is in the nature of a hypertonic solution and produces no toxic symptoms. The author comments on the peculiar characteristic of the audiometric picture in labyrinthine hydrops, which combines the appearance of transmission deafness with that of a perceptive involvement; it was this which led Mygind to term it "a hypoacusis of altered transmission of the internal ear."

Falconnet, Portmann, and Alavoigne[31] contribute a careful experimental study of auditory fatigue tests as a means of detecting those individuals who are particularly susceptible to deafness as a result of working in noisy surroundings. They discuss briefly the divergence of views on methods of performing such tests which exists in the literature. Their own method involved the use of white noise (for three minutes at a fixed intensity of 100 db.) through special earphones in sound-proof rooms. Groups of normal persons, 100 between 17 and 20 years, and 38 between 19 and 25 years, as well as groups of persons who had worked for varying periods in noisy industries were tested; the latter were sorted carefully as to the length of previous exposure to industrial noise. The temporary reduction from previously ascertained thresholds as well as length of time necessary for recovery were noted in each case. Results were tabulated in percentages. The findings were interesting in that they showed not only an average time for recovery but also both unusually susceptible persons as well as some others who suffered no temporary hearing loss at all. This finding, which is in accord with the results of other investigators, could not, however, be correlated with the hearing status noted among those experimental subjects who had worked for a considerable period in noisy industries. The authors conclude that at present we do not know the relationship between the temporary hearing loss which occurs as a result of fatigue tests and the permanent hearing loss which takes place in many persons after working for a considerable period in noisy surroundings. They believe that we are not justified in barring a person who shows abnormal auditory fatigue from working in noisy surroundings on the strength of this test alone. They state that the essential prophylaxis against noise continues to involve rational acoustic treatment of the machines which are responsible, audiometric screening during the first months of employment, and the use of ear defenders.

Luscher [32] discusses recruitment in cranial trauma as indicated by the difference limen test. He cites several cases of inner ear hearing impairment due to this cause in which recruitment was invariably absent in contradistinction to hearing loss due to acoustic trauma where recruitment is almost invariably present. After discussing the hypotheses for this occurrence which are advanced by other authors, he advances the opinion that the reason for the absence of recruitment following cranial trauma lies in the psychic state produced by the accident in the patient.

Manzini, Marenzi, and Teseo [33] report on the results of a hearing survey of 59,850 school children in the elementary schools of Milan. They review the results of similar studies in other countries and cities as well as the best methods used. The latter they list as (1) the fading numbers test; (2) the Massachusetts hearing test, and (3) the sweep test check. They decided on the latter as the most practical, using a tone of 20 db. intensity through all the frequencies from 256 to 8192 but beginning with 1024 up to the highest frequency and returning to 512 and 256 at the end. The total number of defects was 3075 out of the 59,850 children examined, giving a percentage of 5.1%; these were divided into 2415 slight, 540 middle, and 120 severe cases. The authors consider the prophylactic and therapeutic possibilities for each group of hearing losses observed and conclude that group audiometry in school children has great clinical, social, and pedagogic importance.

Shimizu and his co-workers [34] made a study of psychogalvanic skin resistance audiometry. They studied various conditions, such as measuring methods, the confidence limit, individual differences, etc. They found that a condenser discharge current is most useful as the second stimulation for establishing a conditioned reflex. Also the Wheatstone bridge type of circuit is most suitable for measuring the psychogalvanic skin resistance. Both the tester and the patients require much patience since the test and measuring takes a long time. Fatigue alone very often causes a failure of the test. The real threshold of hearing is very difficult to obtain by this test. The test, however, is worth while because it provides data for confirming deaf-mutism and for judging the residual hearing in each sound area—high, middle, and low—in children with various diseases of the ears.

Gibbons and Winchester [35] describe a new objective delayed sidetone test for detecting uniaural functional deafness. Seventy cases of organic uniaural deafness were studied to evaluate the validity of the test. The test consists of first obtaining organic thresholds, including speech reception for the better ear. The subject is then screened to establish his ability to read. Instructions are given regarding the conduct of the test. A sensation level of 60 db. of delayed sidetone is presented to the better ear at the same time a masking noise level of 80 db. is delivered to the apparently poorer ear. The subject is then told to read the test material, which should consist of at least 500 syllables of easily read prose. The elapsed reading time should be measured. The above conditions are then reversed, the sidetone presented to the poorer ear and the masking sound to the good ear, and the subject is again timed reading the same material. If the time difference is less than 10 seconds, a functional element is indicated. A difference of more than 10 seconds between reading times is strongly indicative of an organic uniaural deficit. Dramatic differences in speech production between the two test conditions can also suggest a difference in thresholds even in the small percentage of cases presenting time differences of less than 10 seconds. Clinical utilization of these testing techniques is feasible in standard audiology clinics. The results are objective and their interpretation is easily achieved.

Winchester and Gibbons [36] investigated the relative effectiveness of several modes of delayed sidetone presentation, namely binaural, uniaural, and uniaural with contralateral masking. One hundred sixty

normally hearing adults were tested with these three different modes of delayed sidetone presentation. They found that the binaural mode of presentation is very effective and should be used only when it is not necessary to test ears separately. The uniaural mode of presentation without contralateral masking is the least effective and should not be used as it will yield inaccurate test results. The uniaural mode of presentation with contralateral masking is as effective as the binaural mode of presentation and can be used to test ears separately.

Larr and Leamer [37] discuss the problems confronted in the identification of nonorganic hearing loss, particularly as it applies to children. They feel that nonorganically deaf, mentally deficient, or aphasic children should not be absorbed into the schools for the deaf unless these schools are adequately equipped to handle these specialized problems. The ideal program for identifying organically deaf children and eliminating other possible anomalies would involve the cooperation of several specialties functioning in a well-coordinated center under medical supervision. Provision should be made for the services of an otolaryngologist, a psychiatrist, a neurologist, a speech pathologist, an audiologist, and a psychologist. Those who have the responsibility for pre-entrance examinations of deaf children to deaf schools should consider the following conditions which might resemble functional hearing loss: mental retardation, high-frequency hearing loss, brain injury, hearing loss of a psychological nature, serous otitis media, severe tinnitus, and negativism. Other conditions which might present some problem are the early fluctuations of otosclerosis, faulty mandibular articulation, and emotional disturbances.

Harbert [38] studied the effectiveness and limitations of white-noise masking. Eighty decibels of white-noise masking re normal threshold is the loudest subjectively acceptable level of masking for clinical use in both air- and bone-conduction measurements. This level produces an overmasking of about 5 db. by bone conduction for frequencies 256 and 512 and does not affect the other frequencies. Frequency 256 is the most difficult to eliminate by masking and requires 15 db. more masking than the other frequencies to get equivalent effective masking. For the speech frequencies 30 to 35 db. of masking is ineffective for eliminating crossed hearing by bone conduction. These statements apply only when the ear to be masked has normal or near normal hearing. It may also apply to ears with nonrecruiting deafness but not to ears with conductive deafness.

Siegenthaler et al.[39] substantiate, with their study, the finding of increased sensitivity for bone-conducted sound with occlusion of the external auditory meatus. They find, however, that occluding the meatus does more than reduce interfering noise. Their experiments demonstrated the occlusion effect in very quiet environments and showed a residual occlusion effect when the masking noise was accounted for. Two clinical applications of increased bone conduction with occlusion of the external auditory meatus are indicated. First is the possibility that if a standard occlusion can be used and if the occlusion effect can be quantified precisely, an occluded bone-conduction threshold could be obtained and could be corrected for the occlusion effect. Secondly, the occlusion effect might be put to use to improve perception of some frequencies for bone-conduction hearing-aid oscillation which have comparatively poor output.

Harbert et al.[40] made a study to determine the difference in response to masking of a normal ear and of one with experimentally induced conduction deafness as measured by crossed bone conduction. They found that 80 db. re normal threshold of white noise masking, which can for clinical purposes eliminate a normal ear, is inadequate to eliminate a conduction-deafened ear while the opposite ear is being tested by bone conduction. There seems to be no definite correlation between the change in

effective masking and the degree of conduction deafness induced with a wax plug in the external auditory canal. In this type of deafness even a small impairment requires a well-marked increase in masking intensity if the ear is to be masked out. Clinicians should be aware of the limitations until a more effective way of masking sound is used.

Corso[41] made a study to determine the discrimination loss for speech from normal ears with the C. I. D. Auditory Test W-22. This test consists of 200 monosyllabic words divided into four lists of 50 words each. Each of the lists has been phonetically balanced. The words were selected in part from the Phonetically Balanced Word Lists of the Psycho-Acoustic Laboratory, Harvard University. Data were collected on both right and left ears of 139 subjects, aged 18 to 24 years, having normal otological findings, a maximal pure-tone loss of 5 db. at 500 to 2000 cps inclusive and a maximal hearing loss of 20 db. on C. I. D. Auditory Test W-22. The mean discrimination loss for speech for all ears combined was 2.32% with a standard deviation of 1.80%. These results for untrained listeners are in close agreement with results reported for experienced listeners using the same test. (This test together with an appropriate set of instructions is distributed by Technisonic Studios, 1201 S. Brentwood Blvd., St. Louis 17.)

Epstein and Schubert[42] have made a study of reversible auditory fatigue resulting from exposure to a pure tone. Part I of this paper is a report of an attempt to determine the critical fatiguing intensity for a 4000 cps pure tone of three minutes' duration as measured by poststimulating auditory fatigue. They used as measures the amount of threshold shift, length of recovery time, intensity of the exposure tone at the half-octave shift of the frequency of maximal fatigue, and the amount of recruitment present after stimulation. The results of this study indicate that a sensation level of 80 db. approximates a critical fatiguing of 4000 cps for the following reasons: Sensation of the 4000 cps exposure tone greater than 80 db. appeared to produce a significant change in the fatigue pattern as delineated by amount of threshold shift and recruitment factor. Also, for stimulating tone levels greater than 80 db. the frequency of maximal fatigue shifts from one-fourth to one-half octave above the stimulating tone frequency.

Glorig and House[43] present a new concept in auditory screening: The need for rapid auditory screening tests to measure the hearing of large numbers of persons is more urgent now than ever before. This applies particularly to schools, and military and industrial testing programs. The usual screening methods are not completely suitable for any of these three situations. Investigations of large numbers of audiograms demonstrated that in approximately 99% the hearing loss at 4000 cps was as great as or greater than the loss at any other frequency. Several studies were conducted on this basis and all confirmed this finding. As a result, the authors recommend that testing at 4000 cps be used as a screening test in schools, industry, military forces, and in doctors' offices. It has the advantage of saving time, making it possible to do the test in any ordinary quiet room, and requiring an inexpensive lightweight and very durable instrument.

House and Glorig[44] recommend that testing at 4000 cps be used as a screening test in schools, industry, the military forces, and in doctors' offices. This is based on investigations of a large number of audiograms, which revealed that in approximately 99% the hearing loss at 4000 cps was as great as the loss at any lower frequency. Other studies confirmed this finding. This screening method has many advantages: It requires only a few seconds to complete; it can be done in the ordinary quiet room, as an office, first-aid room, etc.; the instrument used is very inexpensive, is lightweight, and very durable.

A study of the repeatability of the 1 db. step pulsed-tone thresholds in normal hear-

ing persons was made by Eisenberg.[45] It is sometimes desirable to obtain pure-tone measurements in 1 db. steps in establishing norms for the investigation of certain psychoacoustic functions. Her findings showed that under controlled conditions pulsed-tone thresholds measured in 1 db. steps have good repeatability.

Quiggle, Glorig, et al.[46] present a method to predict hearing loss for a certain kind of speech, using pure-tone thresholds. This is a step toward an understanding of the pure tone-speech relation. It is not a means for predicting hearing loss for speech in general. This study deals with spondee speech reception thresholds. A multiple regression equation was derived to predict W-1 spondee speech reception thresholds from pure-tone thresholds. It is based on audiometric data obtained from 20- to 29-year-old males who participated in the 1954 Wisconsin State Fair Hearing Survey. The pure-tone thresholds were measured with standard audiometers, and the spondee speech reception thresholds were measured with a calibrated speech audiometer and Central Institute for the Deaf Recorded Auditory Test No. W-1. The equation based on pure-tone threshold measured at 500, 1000, and 1500 cps is

$$X'_s = 6.92 + 0.22X_{500} + 0.35X_{1000} + 0.21X_{1500}$$

When the 1500 cps threshold is included in the equation, all of the thresholds for higher frequencies have partial correlations that are not significantly greater than zero. The standard error of estimate for predictions for hearing loss for speech for this group of 20- 29-year-old males is 5.0 db. The predictions for groups of older men is somewhat larger, 6.3 db. for 30 to 39, and 10.3 db. for 60 to 69 years of age. Other forms of the regression equation were derived with other combinations of pure-tone thresholds. The computed weight of a given pure-tone threshold was different for each combination of thresholds considered.

Kodman Jr.[47] discusses the relationship between speech discrimination and pure-tone thresholds in hearing loss subjects. He paired two arbitrarily selected samples of hard of hearing adults by equating their pure-tone thresholds at 500, 1000, and 2000 cycles. A prediction of the speech discrimination loss from the pure-tone audiogram would have been in error for the older adults. Because of these findings he feels that speech discrimination function should be measured directly by standard speech audiometry in preference to a prediction based on pure-tone thresholds. There is need for a large-scale study of speech discrimination as a function of age in the population. Hearing and fittings should be made on the basis of free field speech audiometry rather than predictions based on the slope of the pure-tone thresholds where a speech discrimination loss is present.

Calearo and Lazzaroni[48] studied speech intelligibility in relation to the speed of the message. In the normal subject the impairment of discrimination due to an increase of the syllabic rate per minute is almost completely neutralized by a simultaneous slight increase of intensity. However, in subjects showing alterations of the auditory path and centers with a normal hearing function the articulation thresholds and curves for variously accelerated messages are considerably different from normal. The impairment of the function of superior association and correlation mechanisms results in a lengthening of the reaction time of auditory paths and centers which leads to an impairment of intelligibility of accelerated speech. In cases of pathology of the auditory cortex the test with accelerated speech may further allow a specific localization of the site of the lesion. Owing to the prevalently contralateral projection of each cochlea the hearing defect is greater in the ear opposite the lesion.

Pierangeli[49] contributes a long article on the classification, pathology, and development of deaf-mutism. Modern methods for diagnosis and treatment of the different grades of deaf-mutism are discussed, and

representative audiograms of both congenital and acquired forms are given. The article does not lend itself to abstracting.

Panconcelli-Calzia [50] believes that the determination of speech perception exclusively is a utopian idea. This is a review of various methods of auditory testing. His conclusion is that only a senseless series of sounds leads to a true testing of auditory capacity for speech perception. Neither pure-tone audiograms nor tuning-fork tests warrant conclusions regarding speech perception. The ability of selective combination exerts considerable influence on test results as soon as meaningful words or a coherent text are used.

Calearo [51] describes a new test for the diagnosis of simulated unilateral deafness based upon the difference of intelligibility of a message periodically switched from one ear to the other or periodically interrupted in one ear. The ordinary audiometer coupled to a Bocca's periodical interrupter is used. The switching or interruption rate is always on a 50% on-off ratio and can be varied from a minimum of 2 to a maximum of 12 per second. The speech material used was several lists of meaningful short sentences recorded at an average speed of 85 words per minute and presented to the subject through headphones. The intensity used for the alternated message is about 30 db. higher than the hearing threshold in the healthy ear. If one ear is deaf, the normal ear listens to a periodically interrupted message and the discrimination gradually rises from zero to 100% as the switching rate increases from two to eight or nine per second. If discrimination approaches 100% when the switching rate is below three per second, it clearly indicates that the allegedly deaf ear can perceive the fraction of the message interrupted in the hearing ear and shows that the ear is not deaf. The intensity level at which 100% discrimination is obtained by a switching rate below three per second will roughly indicate the amount of hearing loss in the simulated deaf ear.

Industrial or Occupational Deafness

Bernabei [52] made a study of 252 workers employed by the "Largerello" electric works. Most of these were examined by means of group tests using a modified Massachusetts Hearing Test, but a number were given individual audiometric examinations; careful examinations of the ears, nose, and throat were also made. The average noise level was 102-104 db. Workers were classified as to age and length of service, and tables are presented showing average losses for 500, 1000, 2000, 4000, 6000, and 8000 frequencies as compared with the years of exposure. An interesting composite audiogram is attached. Screening for abnormal sensitivity to noise and the use of suitable ear plugs which attenuate 20-30 db. of noise are recommended as prophylactic measures.

Pierangeli and Ghirlanda [53] contribute a long article on the occupational deafness of railroad personnel in Italy. The investigation was classified according to three types of workers: machinists and firemen, traveling and operating personnel, and railway station personnel. There is a complete summary of existing literature on hearing losses in workers of this type, as well as details of their investigation, including noise-level measurements under different conditions. The latter varied from 80-85 db. to as high as 114 db. In the first group the results of the investigations indicated the existence of frequent lesions, generally of the perceptive type, without reaching, in most cases, a considerable degree but slowly growing more intense in the course of years. In the second group, lesions of upper tones are more pronounced, spreading later to middle and low frequencies. In the third group lesions of the inner ear are also present but to a slighter degree than in the others. Prophylactic measures are suggested; these involve careful audiometric screening of personnel, redesigning of equipment to eliminate as much noise as possible, and shifting of workers with definite hearing defects to less noisy positions.

Frugoni [54] discusses the audiometric evaluation of hearing loss with particular reference to the Italian military forces. He observes that the tables for disability in the Italian Army as well as in some others are based on ability to hear the voice at a given distance in meters. This method, while based on the fact that the human voice is the most physiologic stimulus, is also open to considerable variation; even recorded tests require a high degree of cooperation. He reviews the percentage methods for determining ability to hear speech from audiometric records and states that while universally accepted they do not fit the Italian language. With this in mind he has constructed a table based on the methods of Fowler and of Pellegrini which translates audiometric losses for the frequencies of 500, 1000, 2000, and 4000 into corresponding metric distances for hearing Italian words. This method can be used as a check on spondee tests. He also discusses the screening of personnel who are abnormally sensitive to noise and the use of ear defenders where excessive gunfire prevails.

Schulthess [55] studied the effect of noise on the hearing of control subjects and the effect on workers constantly exposed to noise. A method developed by Rüedi for testing individual susceptibility was employed. The Békésy audiometer is used. Noise stimulation deafness is characterized at its onset by a wide audiometrical dip encompassing two octaves between 2000 and 8000 cps, with the maximum between 4000 and 6000 cps. A test subject operating the Békésy audiometer himself at the threshold of 1000 and 4000 cps alternatively will show the following prestimulatory and poststimulatory changes of the auditory function when noise reaches damaging intensity. There is a narrowing of the difference limen of the test tone 4000 cps resulting in a reduced amplitude of the Békésy curve. There is a delayed poststimulatory recovery of the test tone 4000 cps to its original threshold. The writer confirms the existence of individual differences in sensitivity of the human ear to noises of high intensity. Critical intensities of more than 90 db. indicate good resistance to noise and those of less than 90 db. show susceptibility to stimulation deafness. Those subjects found sensitive to noise influence required a longer recovery time than the resistant persons. After administration of 75,000 I. U. of vitamin A daily the test subjects definitely recovered more rapidly from their stimulatory deafness, an average of four to six hours sooner than that of the control subjects without vitamin A. Similar investigations with shift workers at a cement mill confirmed these findings.

Kobrak and Domeier [56] describe a machine designed for automatic self-recording mass audiometry which can be used for routine and follow-up testing in industry. The machine briefs the examined person first, gives pure-tone and speech tests, is able to test each ear separately by headphones over each ear, is able to employ some malingering tests by giving the tone to either ear by repeated switching from one ear to the other, and records all findings automatically on paper. As many as 50 people can be tested at one time. The time for the test is 10 to 15 minutes. The tone source is a tape recorder which is synchronized with a certain paper blank so that the arrangement of the squares harmonizes with the test tones. The paper recording can be passed in the individual's health record. This machine eliminates the two objections which industry has to routine ear testing, namely, time and the need for trained audiometric technicians. Noise protection is provided partially by the earphones. If this is not sufficient, then some sound treatment of the test room should be done.

Glorig [57] presents an industrial testing program. Testing employees' hearing is a necessary part of any preemployment routine physical examination program. Routine preplacement and periodic follow-up audiograms should be made on all employees, especially those who may be exposed to excessive noise. Industrial tests need only record the auditory threshold by air con-

duction. The recommended frequencies are 500, 1000, 2000, 3000, 4000, and 6000 cps. Single frequency, 4000 cps screening tests can be made as follow-up tests. Experience has shown that most of the change in the auditory threshold as a result of noise exposure develops at 4000 cps. It is reasonable to assume that if there is no change at 4000 cycles there will be no change of any significance at any of the frequencies lower than 4000 cps. However, when the loss at 4000 cps is greater than 50 db., this frequency will not accurately reflect the change at other frequencies. Glorig feels that it is imperative that the testing be done under medical supervision.

The audiograms of 1049 male employees of a manufacturing plant have been utilized by Pell[58] to study the relationship of occupational noise exposure to five-year audiometric changes, taking into account age and degree of hearing acuity at the onset of the study period. Analysis of the data shows that hearing acuity of persons with normal audiograms may be affected by five years of subsequent exposure to a steady-state occupational noise ranging from 75 to 90 db. Hearing acuity of persons with moderate high-tone loss, Stage 1, may be affected by five years of subsequent exposure to a steady-state occupational noise of 85 to 90 db. but not by noise between 75 and 84 db. The hearing acuity of persons with severe high-frequency loss, Stage 2, is not affected by five years of subsequent exposure to steady-state occupational noise up to 90 db. Those employees exposed to noise stress who do advance over a five-year period usually advance only one stage; only a small percentage advance more than one stage. High-frequency hearing loss begins rapidly and proceeds more slowly in its advanced stages. The damaging effect of noise stress diminishes with each successive initial stage. Persons 45 years and over are more susceptible to traumatic hearing loss than persons below 45 years. Noise safety criteria must also take into consideration the number of years of exposure, the kind of hearing loss the noise produces, the age and degree of neural deafness of the exposed personnel, and variation among persons in their susceptibility to traumatic hearing loss.

Fox[59] stresses the importance of and growing interest in occupational hearing loss. The otolaryngologist should become interested in and study the many facets of this problem. He should consult with the industrial hygienist, the plant physician, and other necessary personnel. In medicolegal evaluation he should be objective, impartial, and scientific. In this way he can play a vital role in this problem. A chart and method for determining hearing loss and hearing handicap used in Wisconsin accompanies the paper.

Malingering

Stützer[60] has devised a test to uncover unilateral malingering deafness or hard of hearing. The method consists in covering tightly one ear with the air-conduction receiver, while tones are sent over the bone-conduction receiver into the room. The bone-conduction receiver lies on a table in front of the patient. The tones emitted from the bone conductor are first heard by the other ear. The patient is under the impression that this is an examination of *that* ear which is covered with the earphone. The procedure is as follows: (*a*) First, patient's history is taken and local findings noted. (*b*) Whisper and conversational voice tests are performed. (*c*) The stimulation test is now proceeded with, *before* threshold audiogram tests are done, in the following manner:

On a small table in front of the audiometer are placed the air-conduction receiver, the bone-conduction receiver, and the masking receiver. The bone-conduction receiver with its sound-producing membrane upward lies between the two, and all are in front of the patient. The patient then puts the air conductor to the so-called healthy ear, which he is told is being tested. In the meantime tones (c′-fis^5) are being sent through the bone conductor into the room. The patient

is asked to indicate when and how long he hears. Thus the apparently sick ear is being tested. Now the procedure is reversed. The apparently sick ear is covered with the air conductor, and the patient is told that this ear is being tested. Malingerers will tell that they hear nothing, and are thus uncovered. After this the usual routine threshold audiograms are done.

In evaluating the testing results one has to take into consideration the distance of the patient from the bone-conduction receiver. The tested ear should not be farther than 1 meter away from the source of sound.

This method is meant for the daily practice as a first test, especially when malingering deafness is suspected.

Rehabilitation

A complete list of the hearing centers of the United States was compiled by Downs and Carson [61] from questionnaires sent to the centers the latter part of 1956. This article lists a total of 145 centers grouped according to states, giving the name of the controlling school or group under whose auspices each center is being run, the administrative head, and the medical supervisor. A table accompanies this article giving the type of personnel, their special training, and the type of service which each center has to offer.

Saltzman [62] discusses various factors which should be considered in determining a person's ability to learn speechreading. He describes Keaster's plan of study for correlating the lipreading ability with specific psychological aptitudes. Lipreading instruction is most successful in the young. Phenomenal speechreaders predominate in congenital and postmeningitis deafness. The person with tinnitus has more difficulty with visual speech because the tinnitus is a drawback to his concentration and attention. Likewise in Ménière's disease the sounds reaching the supersensitive and distorted inner ear become a distractive factor. Slow cerebration, as at times found in hypothyroidism, arteriosclerosis, and anemia, may augment the effects on hearing impairment. Our advances in otologic diagnosis make it possible to single out the hard of hearing person with special discriminatory difficulties. As the span of life increases, a greater number of people with slow cerebration will appear. These will require special medical as well as otologic rehabilitation.

Anthony and Garaway [63] suggest the use of binaural-time delayed auditory stimuli for rehabilitation of the nerve deaf patient who has poor discrimination. This is achieved by using two separate systems of sound amplification with one sound source and varying the time relationship of the output of one receiver to the other by 1/400 to 1/25 of a second. With orthodox electrically amplified binaural hearing overamplification in one ear decreases the discrimination ability of the other ear. With the delayed stimulus effect, the ability to discriminate does not seem to be impaired by changes of loudness, and the ears are able to take the increased intensity without discomfort. By delaying the sound to one ear we cause the discharge of impulses to the cerebral cortex at a different time from each ear for a given sound without subjective confusion. This is the key to the possible application of the phenomenon to rehabilitation of the nerve deaf patient. Further evaluation of this is now in process.

Wildervanck [64] examined 15 sets of presumably normally hearing parents and their 32 congenitally deaf children who probably had recessive hereditary deafness, in order to see whether these parents were detectable as heterozygotes. Heterozygotes are people who have a single gene for a certain quality. In cases of recessive heredity one may expect that the parents, with good hearing, of the deaf child are heterozygotes for the gene "deafness." Related parents are more frequently both heterozygous for some genes, such as deafness, than are nonrelated parents, as they both have a great chance to inherit the gene from one of their common ancestors. Threshold audiograms of these parents and their deaf-mute children

are presented. The parents turned out not to be detectable as heterozygotes. The importance of consanguinity is emphasized.

Bergman [65] discusses the rapidly expanding and experimental interest in binaural hearing, which demands bold new thinking for clinical application. The claimed advantages of two-eared hearing over the one-eared is localization, selectivity, better discrimination in quiet and in noise, ability to identify common sounds, ease of listening, and better quality of sound. He made a preliminary study of localization as a function of intensity, using special equipment. He found that localization, and very likely other aspects of binaural hearing, does occur in persons with ears which are markedly different from each other, provided the sound is above the audibility threshold of the poorer ear. He suggests we study some of the disturbing auditory difficulties still suffered by patients who have had some improvement of hearing in one ear either from surgery or the wearing of an aid. It is apparent that a new battery of tests will have to be developed to quantify the advantages of binaural hearing.

Whitnall [66] states that few deaf children are totally deaf. It is necessary to know something of the development of speech and hearing in the normal child, and to appreciate the period of "readiness to listen" preceding the period of readiness for speech. Very few children are totally deaf. Most have some hearing that can be developed by auditory training. A hearing aid and hearing environment are necessary. Even children with no response at the first test may be found to have usable hearing after adequate auditory training.

The aim of auditory training is to reproduce for the deaf child, so far as possible, the listening opportunities of the hearing child.

The deaf child should be trained to hold his own in the normal hearing world.

Miscellaneous

After citing seven different theories of direction of sound source and their various supporters, and commenting on them, the authors, Kitigawa and Shintaku [67] present their technique, which is to have the subject, with eyes shut, point the index fingertip toward the sound source and have the fingertip approach the skin surface, keeping the tip and the sound overlapped. Many illustrations of the head show the points indicated where the sound appears to be, including bone-conducted sound, and sound under water. Direction perception appeared to be monaural on the lateral sides and binaural in front and at the back.

Maraist and Hutton [68] subjected 15 stutterers to reading aloud a set of five passages under five levels of masking noise. Errors and duration of passages decreased as masking level increased from 0 to 90 db. The rate of words per minute increased as masking level increased. Under 90 db. masking noise subjects' mean rate of 144 words per minute approximated normalcy.

Forty-eight patients who had been subjected to electric shock treatment revealed no change in hearing acuity or in other functions of hearing, according to examinations made by Flattorp, Wille, and Eitinger.[69]

Street [70] examined 90 aphasic patients in a Veterans' Hospital, aged 19 to 70. Tests were made for auditory comprehension, reduced attention span, and communication difficulty. The most difficult cases were those with extensive brain damage. Patients with impaired auditory retention can benefit from increased amplification. Patients with impaired auditory attention with hearing loss at 1000 and 2000 cps can frequently benefit from amplification. If aphasic patients with hearing losses fail to benefit from a program of lipreading and ear training, a hearing aid should be tried.

Speech specialists may be interested in the attempted investigation by Williams and Bangs [71] of the relationships between speech discrimination and other audiometric data, namely, pure-tone threshold losses at commonly used intervals; cochlear reserve at recruitment and speech reception threshold.

They also took into consideration what they called the index of masking, an inferred estimate of masking of speech by other speech sounds.

Audiometric investigation of the tactile threshold of the mastoid is recommended by Oeken.[72] The tactile threshold of the mastoid was investigated by audiometric bone conduction in unilateral and bilateral deafness. Values below the tactile threshold curve should be regarded as tactile perceptions. Disregard of this may lead to diagnostic errors.

Sataloff and Menduke[73] studied a group of elderly people, 94 men and 101 women between the ages of 64 and 93, who had no history of significant acoustic trauma or disease. This study was for pure tones only and did not reflect an elderly person's ability to hear and understand speech. Their results show that little of the variation in hearing loss can be accounted for by age differences after 65 years of age. The results were consistent with the thesis that little additional hearing is lost after age 65. Long-term follow-up studies, however, are needed before this can be advanced as a definite conclusion.

Under the title, "Theoretical Note on Some Types of Psychogenic Deafness," i. e., central, conductive, perceptive, Getz[74] states that psychogenic deafness may take on the appearance of conductive, perceptive, or central types. Manifestly the diagnosis depends on recognition of the underlying psychogenic cause. (He does not go into the method of recognition of this cause to any extent.)

Cawthorne[75] says it is difficult to differentiate chronic adhesive otitis from otosclerosis. It may even cause stapes fixation. Marked changes in the tympanic membrane and history of inflammation are useful (Abstracter's suggestion: also failure of pneumatization of the mastoid). Fenestration alone is not successful in these cases. If the stapes is mobile, another fenestra on the vestibular side is likely to cause a disturbing condition known as the Tullio phenomenon. Loud sounds cause dizziness. Author suggests more attention to the round window. In the discussion, L. B. Thorburn, quoting Wullstein, suggested that, if the round window could not be seen, the impulse transmitted to it can be observed through a drop of fluid over the niche of the round window.

An assessment of the functional state of the deranged middle ear with an electrical acoustic probe is recommended by Bennett.[76] A description of the probe and its diagnostic application is given. Author proclaims its usefulness in selecting the proper tympano-plastic procedure.

Kennedy[77] made a study of 433 school children to show that hearing is a maturational process without any significant sex or laterality differences. This maturation phenomenon does not present a straight-line pattern but rather only spurts and plateaus. There is a significant difference between the 6- and 8-year-old groups and somewhat less difference between the 8- and 15-year-olds. The 10-year-old boys seem to have somewhat less acute hearing than the 8-year-olds. However, the 10-year-old girls exceeded the 8-year-olds. Both boys and girls showed a drop at the 12-year level. This would mean that the upper elementary grades may be a period of particular concern with regard to potential loss of hearing. Different standards of hearing acuity are needed for the evaluation of different age groups below the adult level. Certain age groups deserve special attention in planning testing programs because of the higher incidence of loss and the greater severity of difficulty at given age levels. The failure to recognize age differences has resulted in misinterpretation concerning the feasibility and accuracy of hearing tests with young children.

Saunders[78] reports three cases of conductive deafness due to Pyle's disease (familial metaphyseal dysplasia). All cases showed a marked hyperkeratosis of the facial bones, cranium, and mandible, and all gave deafness as a presenting symptom. The tympanic membranes were normal.

One child had a pure conductive deafness, and the other child showed some evidence of nerve loss of function, as evidenced by a drop in the 1024 and 2048 frequencies, especially for bone conduction. The third patient, the mother of the first child, had a more pronounced deafness with a strong neural component. Deafness is believed due to fixation of the stapes or obliteration of the round window or both. Changes in the skull and facial bones have not been associated with the previously reported cases of Pyle's disease, nor has deafness ever been reported associated with this condition.

Hallberg [79] made a study of 178 cases of sudden deafness of obscure origin. Eighty-nine cases seemed to have a probable vascular explanation. The commonest cause of sudden deafness is thought to be a vascular accident involving the blood supply to the cochlea, labyrinth, or both. There appears to be some evidence to show that patients so afflicted are more susceptible than the average patient to the development of other types of vascular accidents later in life. In addition to a careful examination a complete general examination is indicated, including tests for cholesterol, fatty acids, and total lipids in the blood. If they are high, the possibility of atherosclerosis must be considered.

25 E. Washington St.

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Books

BOOKS RECEIVED

Clinical Enzymology. Edited by Gustav J. Martin, Sc.D. Price, $6.00. Pp. 241, with 5 illustrations. Medical Book Department, Little, Brown & Company, 34 Beacon St., Boston 6, 1958.

***The Esophagus: Medical and Surgical Management.** By Edward B. Benedict, M.D., and George L. Nardi, M.D. Price, not given. Little, Brown & Company, 34 Beacon St., Boston 6, 1958.

Sprachhörprüfmethoden: Grundlagen, Würdigung und Anwendung bei Begutachtung und Hörgeräteanpassung. By Prof. Dr. Med. Dipl.-Ing. Kurt Schubert. Price, $16.45. Pp. 361, with 187 illustrations. George Thieme Verlag, Herdweg 63 (14a), Stuttgart. (American agent—Grune & Stratton, Inc., 381 4th Ave., New York 16), 1958.

Ciba Foundation Symposium: Chemistry and Biology of Mucopolysaccharides. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Maeve O'Connor, B.A. Price, $8.50. Pp. 323, with 48 illustrations. Little, Brown & Company, 34 Beacon St., Boston. Published in England by J. & A. Churchill, Ltd., 104 Gloucester Place, Portman Sq., London, W. 1, 1958.

BOOK REVIEWS

Practical Allergy. By Coleman Harris, M.D., F.A.C.P., and Norman Shure, M.D., M.S., F.A.C.P. Price, $7.50. Pp. 471, with 25 illustrations and 16 charts. F. A. Davis Company, 1914-16 Cherry St., Philadelphia 3, 1957.

This volume is based on lectures and demonstrations given to students in the College of Medical Evangelists, Los Angeles. It is, therefore, elementary, and is aimed for use by those practitioners (the G. P.'s) who are not specialists. Chapter 7 deals with "Nonpollen Causes of Inhalant Allergy" such as house dust, animal effluvia, orris root, kapok, fungi, and molds, and bacterial allergy so often overlooked or set down as "virus" (Chapter 13).

Major factors are, of course, prevention, diagnosis, and desensitization. The last is highly important, especially in these latter days of rather indiscriminate administration of antibiotics and vaccines. It is possible to induce allergy by unwise or indiscriminate use of remedies—drugs and other agents. Patients and physicians should be alert to such possibilities. The authors have done well in laying the groundwork for better understanding of a diffuse and sometimes baffling subject.

Surgery in World War II: Ophthalmology and Otolaryngology. Medical Department, United States Army. Col. John Boyd Coates, Jr., (MC) U. S. Army, Editor in Chief; M. Elliott Randolph, M.D., Editor for Ophthalmology; Norton Canfield, M.D., Editor for Otolaryngology; Elizabeth M. McFetridge, M.A., Associate Editor. Prepared under the direction of Major General S. B. Hays, Surgeon General, United States Army, by The Historical Unit, United States Army Medical Service. Price, $5.00. Pp. 605, with 149 illustrations including charts and tables). Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., 1957.

This volume is the ninth in a series published by the Medical Department of the United States Army, under the distinguished over-all editorship of Col. John Boyd Coates (MC), with the subeditorship of eminent specialists in the two subjects covered.

The workmanship of the volume is superb, the paper and the binding are of the best, and the illustrations well done. The two pages in color are especially attractive.

It will be of greatest interest to those who served in the Armed Forces during World War II, but almost equally so to nonmilitary readers who will want to know what was done by military surgeons during that war. The Editor for Ophthalmology is M. Elliott Randolph, M.D., and for Otolaryngology, Norton Canfield, M.D.

* Will be reviewed.

In Ophthalmology there are 19 chapters contributed by an equal number of eminent ophthalmologists. To quote from the preface of this section:

"This section on ophthalmology in the history of the Army Medical Department in World War II is based on the work and experience of the chiefs of the Ophthalmological Branch in the Surgeon General's Office, the overseas consultants, and medical officers concerned with problems related to the Eye. It is a record of administrative phases and the clinical aspects of ophthalmology, as reflected in the policies and practices concerned with both the specialty and its associated activities, such as the optical and artificial eye programs and, most significantly, the program for the rehabilitation of blinded casualties. Some of the clinical procedures described may seem, a decade later, outmoded and archaic, since newer methods have been developed in the progress of ophthalmology; but the administrative experiences may serve as a basis for the conservation of time and energy in the event of another conflict."

The following headings indicate the contents of these 27 chapters, and it will be seen what a wide and satisfactory coverage is obtained:

I. Administrative Aspects of Ophthalmology in Zone of Interior
II. General Clinical Policies in Ophthalmology in Zone of Interior
III. Administrative Aspects of Ophthalmology in the Mediterranean Theater of Operations
IV. Management of Battle-Incurred Injuries of the Eye in the Mediterranean Theater of Operations
V. Administrative Aspects of Ophthalmology in the European Theater of Operations
VI. Clinical Policies in Ophthalmology, European Theater of Operations
VII. Administrative Aspects of Ophthalmology in the Southwest Pacific and Pacific Ocean Areas
VIII. Clinical Policies in Ophthalmology in the Southwest Pacific and Pacific Ocean Areas
IX. The Rehabilitation of Blinded Casualties
X. The Management of Intraocular Foreign Bodies in Zone of Interior
XI. The Managment of Intraocular Foreign Bodies in an Overseas Ophthalmic Center
XII. Uveitis in Zone of Interior Hospitals
XIII. Retinal Detachments
XIV. Management of Cataracts in Military Personnel
XV. Nutritional Amblyopia in American Prisoners of War Held by the Japanese
XVI. Dark-Adaptation Studies on Night Blindness in Military Personnel
XVII. Visual Disturbances Associated with Head Injuries
XVIII. Plastic Surgery of the Lids and Orbit
XIX. Chemotherapy and Antibiotic Therapy in Zone of Interior

Likewise, from the preface to the Otolaryngology section:

"In what follows are general statements of policy and specific details of important phases of combat otolaryngology. There were so many well-qualified specialists who served that it has been difficult to select all of the contributions which were made. The day-to-day care of the fighting armies required most of the specialists' professional time, and this was cheerfully and continuously available, both at home and abroad. Assignment of medical officers to strategic posts, arranging for regional consultation, and association with colleagues in the countries through which the armies passed was a constant mission of the administrative chiefs.

"The authors of the various chapters were chosen because of their outstanding qualifications and contributions. They, as well as many others, were constantly alert to the possibilities of gaining extra knowledge from their experiences. They were previously, and they continue to be, leaders in otolaryngology in civilian life."

It is not possible to review each individual chapter, but the mention of a few may not be out of place, with the understanding that all merit the commendation of the readers. The headings of the chapters in this section give a condensed idea of the subjects covered:

I. Administrative Aspects of Otolaryngology
II. Clinical Policies in Otolaryngology
III. The Aural-Rehabilitation Program for the Deafened and Hard of Hearing
IV. Narcosynthesis in the Management of Functional Deafness
V. Auditory Impairment Due to Battle-Incurred Acoustic Trauma
VI. Unrecognized Auditory Trauma From Battle Noise
VII. Facial Paralysis in Military Personnel

This reviewer unhesitantly recommends this volume for inclusion in any ophthalmo-oto-laryngologist's library. The price is surprisingly low, and the entire work filled with most interesting and useful information.

A. M. A. Scientific Exhibits, 1957. Edited by Editorial Committee, Council on Scientific Assembly, American Medical Association (various contributors). Price, $15.00. Ppp. 480, mostly illustrations. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1957.

This third volume is a credit to the publishers. The reproductions of the various exhibits are presented with great clarity and make reading and interpretation very easy.

As in the previous volumes, the award winners were chosen, but with apologies to the other exhibitors. As indicated in the preface, this was done because of lack of space, but without any reflections on the caliber or importance of those exhibits that were omitted.

This book is valuable in that the physician can review many of the exhibits at his leisure, study many that he may have missed during the hectic days of the convention, and can obtain reprints and other information on the many subjects of interest. This book is of great value to physicians in every branch of medicine and surgery.

La tonsilla palatina nei suoi lineamenti biologici e fisiopatologici. By Alessandro Fioretti. Price, 3000 lire. Pp. 482, with 94 illustrations, partly in color. Edizioni Deca, Milano, 1957.

This monograph is built around the experimental work on tonsillar physiology and pathology as carried on at the department of professor Michele Arslan in Padova. The latter, in his foreword, emphasizes the importance of a similar synthesis on problems of morphology, physiology, and pathology to arrive at an understanding of clinical problems. The discussion is opened by 36 pages of a historical outline (by Premuda and Fioretti) followed by a chapter on comparative anatomy amply supplied with synoptic charts. The subsequent sections deal with embryogenesis, postnatal evolution and aging, microscopical anatomy, participation in intermediate metabolism, role in "dysreactivity"—allergy, shock—finally, written by Michele Arslan, focal pathogenesis. The bibliography occupies 36 pages, testifying to the broad basis of the study.

In the never-ceasing flow of tonsillar investigations it is desirable to arrive from time to time at a synthesis of the tremendous material. The book is just this and it should be welcomed by the many who are interested in the basic principles around this important topic per se or by those who consider them to be the indispensable groundwork in their day-to-day clinical activity.

Maladies de l'oreille interne et oto-neurologie. By M. Aubry, Professeur agrégé à la Faculté de Médecine des Hôpitaux de Paris, and P. Pialoux, O. R. L. des Hôpitaux de Paris. Price, not given. Pp. 706, with 197 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris 6e, 1957.

This timely book presents a comprehensive discussion of the diseases of the internal ear and neurootology. It is divided in three sections, the first of which deals with anatomy, physiology, and the various diagnostic procedures of the vestibule and the cochlea. There is a detailed discussion of embryology, histology, and the neuroanatomy of these two organs. Under physiology there is a review of the recent experimental work on both man and animals. The various tests, both vestibular and cochlear, are described in detail, giving their significance, importance, and their relation to normal and diseased patterns.

The second section deals with pathologic conditions of a peripheral nature. This includes a chapter on endolabyrinthitis due to trauma, inflammatory conditions, vascular disturbances, and angioneurotic alterations. The problems of the various types of vertigo, deafness, and tinnitus with their differential diagnosis are fully discussed. Noninflammatory conditions—particularly Ménière's syndrome—and the various toxic factors that may produce labyrinthine symptoms of a peripheral nature are presented. Another chapter deals with symptoms that are due to retrolabyrinthine conditions.

The last chapter of the book is devoted to neurootology. Abnormal findings of nystagmus and vertigo are correlated with the cochlear findings and their significance interpreted. Lesions of the central nervous system, both supra- and infratentorially are described, with a detailed description of the vestibular and cochlear findings so that a differential diagnosis can be established. Many of the syndromes have been substantiated by either operative or postmortem findings.

The text is supplemented by numerous illustrations and audiograms which add to the clarification of the subject matter. Also, there is an extensive bibliography. This excellent book, which is presented in a most interesting and logical manner, should be of extreme interest to the internist, otologist, neurootologist, and neurosurgeon.

Year Book of Ear, Nose and Throat and Maxillofacial Surgery (1957-1958 Series). Edited by John R. Lindsay, M.D.; Dean M. Lierle, M.D., and William C. Huffman, M.D. Price, $7.50. Pp. 383, with 96 illustrations. The Year Book Publishers, Inc., 200 E. Illinois St. Chicago 11, 1958.

This volume is a departure from the previous publications in that for the first time it is issued as a separate book devoted to otorhinolaryngology. The scope of the book has been widened and includes many fields of interest, such as allergy, peroral endoscopy, and voice production. A new section for maxillofacial surgery has been added.

The otologic section deals with investigations of the vestibular function, and vertigo, hearing tests and hearing, tubal function and inflammatory diseases of the ears, and otosclerosis.

The next section deals with the nose and sinuses, the mouth and oropharynx and salivary glands, the larynx and neck, the trachea, bronchi, and tracheotomy. Allergy and the viruses are accorded a considerable amount of space.

The maxillofacial section occupies more than one-third of the book.

In recognizing the changing trend in the practice of otolaryngology, the editors wisely placed emphasis on the newer problems that face the specialist.

Recent Advances in Oto-Laryngology. Third Edition. By F. Boyes Korkis, M.B., Ch.B. (N. Z.), D. L. O. (Eng.), F.R.C.S. (Ed.), F.R.C.S. (Eng.), Surgeon and Dean, Metropolitan Ear, Nose and Throat Hospital. Price $12.00. Pp. 438, with 146 illustrations, including 2 color plates. Medical Book Department, Little, Brown & Co., 34 Beacon St., Boston 6, 1958.

Osler once differentiated a textbook from a yearbook by pointing out that the former should present views that have stood the test of time, while a yearbook should record the proceedings of the previous year like the minutes of a meeting. This book stands midway between the two; it assesses, comments on, and draws conclusions from the medical literature, dealing with subjects of topical interest. With the exception of timely historical references, the quoted literature has appeared mostly within the past five years.

The editor and compiler of "Recent Advances in Oto-Laryngology" has resisted the temptation to farm out various chapters. Instead, he has made a personal survey of the entire field of otolaryngology, presenting much information that is useful in clinical practice. Among the more important chapters of this book are those dealing with anesthesia, the deaf infant, acoustic trauma, and the treatment of facial palsy. Surgical advances in the treatment of otosclerosis include pertinent comments on the fenestration operation and mobilization of the stapes, together with their indications, techniques, and results. The long-term results of surgical treatment as opposed to radiotherapy in laryngeal carcinoma are evaluated, and there are sound discussions of Ménière's disease, poliomyelitis, the functions of the nose, tumors, the salivary glands, and the management of esophageal disease. Today's otolaryngologist must necessarily be knowledgeable on a broad front. This book supplies a rich stream of otolaryngological nourishment.

Audiology. By Hayes A. Newby. Price, $6.00. Pp. 342, with 50 illustrations. Appleton-Century-Crofts, Inc., 35 W. 32nd St., New York 1, 1958.

There has long been a need for an authoritative book on audiology which provides a penetrating as well as a general perspective of the subject from the point of view of the professional audiologist as well as the otologist. This book certainly fills this important need. It is a basic reference book and should prove of inestimable value to everyone concerned with clinical hearing studies and deafness. There is a comprehensive chapter on the testing of hearing function with a logical and clear-cut explanation of all the pure-tone and speech audiometric studies as well as the purposes for which they are done. An extensive chapter on special problems in hearing testing is a great contribution for the clinical otologist. The book is particularly helpful in discussing the problems associated with the hard of hearing child and the rehabilitation of the hard of hearing adult. There is an excellent bibliography at the end of the book and very selective references at the end of each chapter. It would be to the advantage of every otologist to read this book from beginning to end.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.
Meeting: Third International Congress of Allergology.
Place: Paris. Time: Oct. 19-26, 1958.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 12-14, 1958.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
President of Congress: Prof. R. David de Sanson.
Secretary of Congress: Dr. Walter Benevides, Caixa Postal 2838, Rio de Janeiro.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Annual Meeting: Palmer House, Chicago, Oct. 12-17, 1958.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 12-17, 1958.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Place: The Homestead, Hot Springs, Va. Time: March 10-12, 1959 (afternoons only).

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: The Homestead, Hot Springs, Va. Time: March 8-9, 1959.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Homestead, Hot Springs, Va. Time: March 10-12, 1959 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, October, 1958.
Annual Meeting: Chicago, October, 1958.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: The Homestead, Hot Springs, Va. Time: March 13-14, 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.
Place: Conrad Hilton Hotel, Chicago. Time: Oct. 12, 1958.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.
Place: Palmer House, Chicago. Time: Oct. 16-17, 1958.

Otosclerosis Study Group

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.
Place: Palmer House, Chicago. Time: Oct. 11, 1958.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.
Annual Meeting: Palmer House, Chicago, Oct. 14, 1958.

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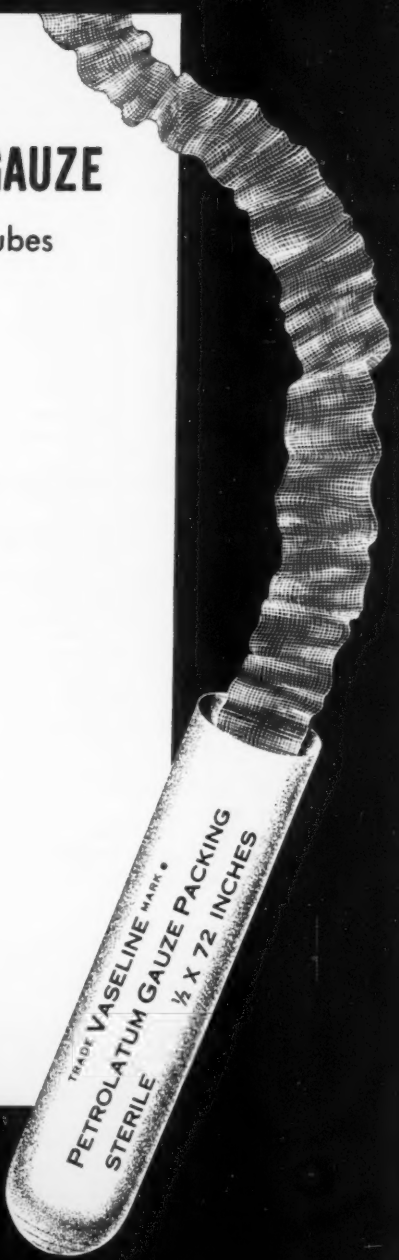

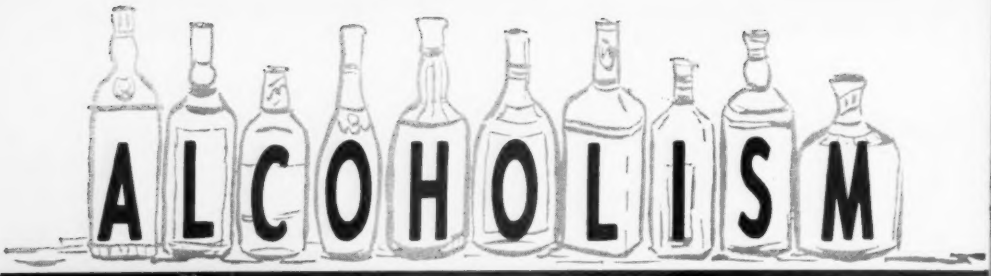

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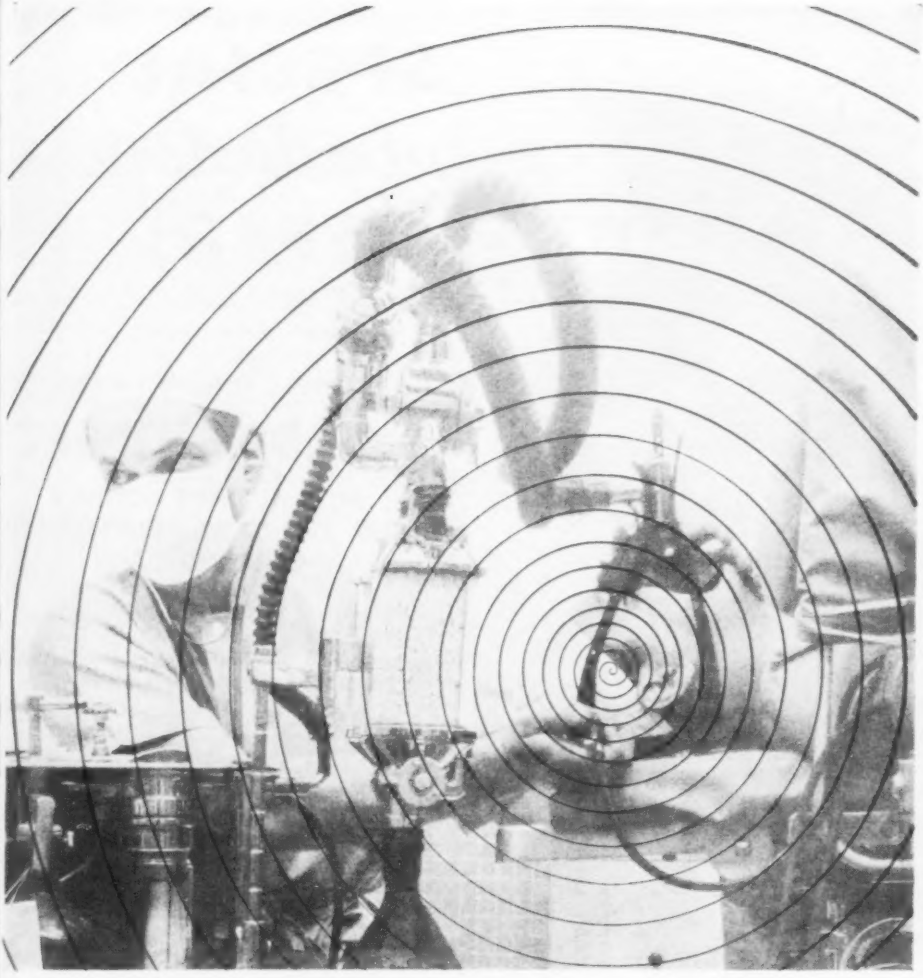

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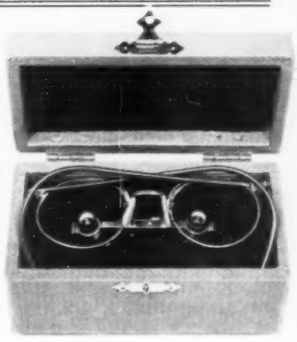

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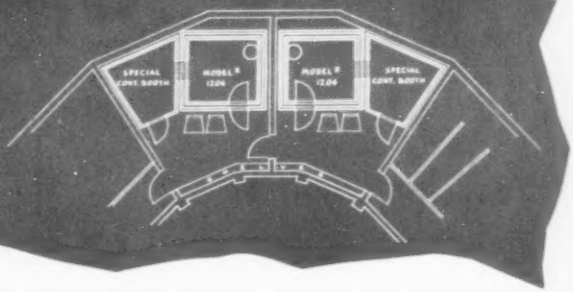

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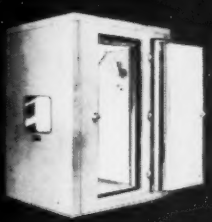

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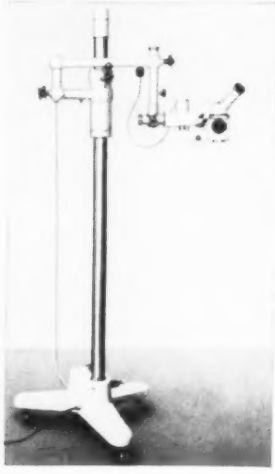

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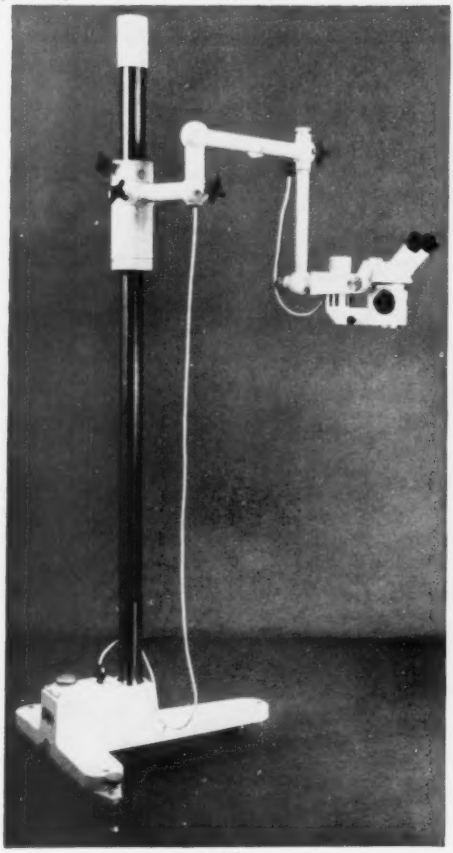

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